SURGERY

VOL. 47 JANUARY, 1960 No. 1

Original Communications

Society for Vascular Surgery

COMMENTS ON THE DISTRIBUTION OF BLOOD FLOW

HARRIS B. SHUMACKER, JR., M.D., INDIANAPOLIS, IND.

(From the Department of Surgery, Indiana University Medical Center)

THE remarkable capacity of the body to maintain a satisfactory internal milieu under ordinary circumstances has long fascinated scientists in general, and those in the various medical branches in particular. With the passage of time observations of this nature have gradually been extended to include not only the more gross adjustments but the more delicate physical, chemical, hormonal, and other reactions as well. We are still, however, in the midst of the quest better to understand these changes, the regulatory mechanisms controlling them, and their role in the body's response to changing external environmental circumstances, internal derangements, injury, and disease.

It is my purpose to speak briefly about certain alterations in the distribution of blood flow, particularly through some of the vital organs. I hope at least to indicate that this may continue to prove a fruitful field for investigation. Because of the limitations of time I shall make no effort to review the literature but instead shall base my remarks upon some studies recently completed or currently being made by my associates* and me. They are based upon satisfactory methods of direct measurement of blood flow in the healthy, lightly anesthetized, well-ventilated, heparinized, mongrel dog. The limitations imposed by the nature of these studies and the species used are recognized.

In 1947 two former presidents of this Society, Michael DeBakey and Alton Ochsner, collaborating with Burch and Ray, published a paper[1] entitled "The 'Borrowing-Lending' Hemodynamic Phenomenon (Hemometakinesia) and Its

Aided by grants from the United States Public Health Service, the Indiana Heart Foundation, and the James Whitcomb Riley Memorial Association, and a contract between the Office of Naval Research, United States Navy, and Indiana University.

Read at the Thirteenth Annual Meeting of the Society for Vascular Surgery, Atlantic City, N.J., July 7, 1959.

*Dr. Hardin is one of my resident staff who is currently working in the Army Medical Research Laboratories, Fort Knox, Kentucky. The others, Drs. Bounous, Heimburger, Jontz, King, Kunkler, Nash, Onnis, Shikata, Su, and Teramoto, are working in my own laboratory. One phase of this study has been published[3]; the others are being prepared for publication. They will contain detailed information omitted from this report which deals only with general considerations.

Therapeutic Application in Peripheral Vascular Disturbances." Those who had not previously appreciated this general principle now had a clear-cut demonstration of it and a plea for its recognition in the management of patients. One might state it very simply by saying that, if the effective blood volume remains the same and the cardiac output is unchanged, any increase or decrease in blood flow to a part of the body is possible only by a concomitant decrease or increase in flow to some other part.

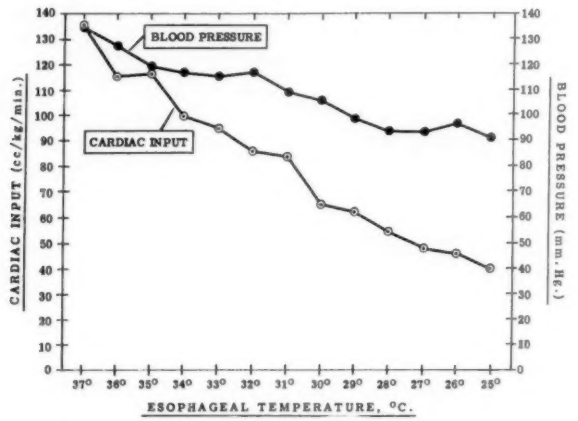


Fig. 1.—Chart showing relationship of body temperature to cardiac input and mean blood pressure.

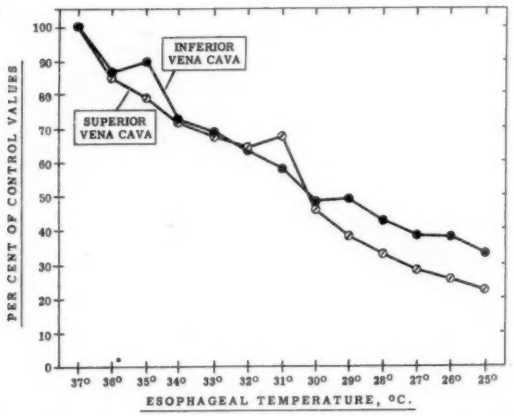


Fig. 2.—Chart showing relative changes in inferior and superior vena caval flows as body temperature is reduced.

First, I should like to discuss some of the alterations in the distribution of blood flow in general hypothermia. If one computes the cardiac output by measuring directly the input into the heart from the superior and inferior venae cavae, one finds that a progressive fall occurs as the animal is cooled (Fig. 1). There is a roughly similar decrease in superior and inferior vena caval return although the superior vena caval flow falls a trifle more than the inferior caval flow as the temperature is lowered below 30° C. (Fig. 2). Blood pressure decreases correspondingly less than the cardiac output, indicating a progressive increase in total body vascular resistance with reduction in body temperature.

At a relatively normal body temperature (36.5° C.) the average renal blood flow is 24.5 c.c. per kilogram per minute; under moderately hypothermic circumstances (30° C.) the average renal blood flow is 12.9 c.c. per kilogram per minute. The portal venous flow falls less, the mean being 30.1 c.c. per kilogram per minute at 36.7° C. and 21.6 c.c. per kilogram per minute at 30° C. Since the cardiac output drops appreciably, the renal blood flow represents roughly the same percentage of total cardiac output in the hypothermic as in the normothermic state and the portal flow a somewhat greater percentage* (Fig. 3). Considering the great general reduction in metabolic requirements brought about by moderate reduction in body temperature, it would seem clear that adequate blood flow to the kidneys and liver is preserved.

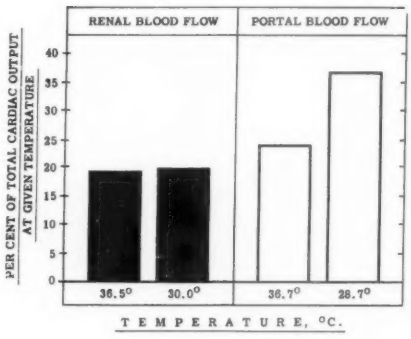


Fig. 3.

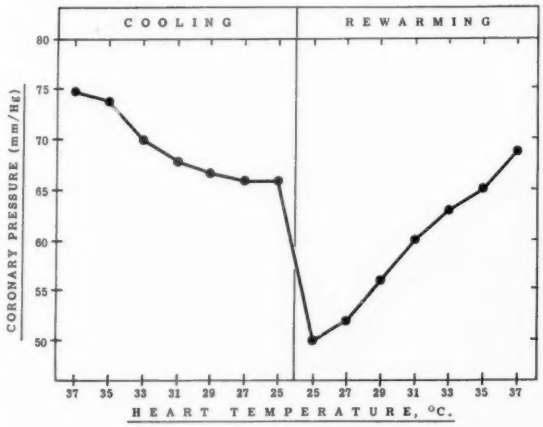


Fig. 4.

Fig. 3.—Chart showing relationship of renal and portal flows to cardiac output under normal and hypothermic conditions.

Fig. 4.—Chart showing changes in coronary pressure with constant perfusion volume as the dog's heart is cooled and then rewarmed while general body temperature is kept at a normal level.

Some interesting observations concerning coronary blood flow have recently been made by Hardin and Scott.[2] During total cardiopulmonary bypass with extracorporeal circulation the body temperature was maintained at normal levels while the heart was selectively cooled to 25° C. and then rewarmed. This was accomplished by drawing off arteriolized blood, passing it through cooling or warming coils and delivering it into the proximal portion of the clamped ascending aorta. Since a constant perfusion volume was used, changes in coronary resistance could be estimated by measuring changes in pressure. A progressive decrease in coronary resistance was observed as the heart was cooled, and an increase as the heart was warmed (Fig. 4). This remarkable change was observed both in animals with normal cardiac innervation and in those with denervated hearts. The increased flow with cooling cannot be explained solely on the basis of better diastolic filling.

*Although renal and portal flows, on the one hand, and cardiac output, on the other, may be compared, it should be pointed out that the former measurements were made under hypothermia induced by body immersion in ice and water while the latter were made under hypothermia brought about by chilling the blood extracorporeally. Our initial differential temperature studies suggest that the two methods differ principally in that a lag in fall of deep muscle temperature accompanies hypothermia induced by extracorporeal cooling of the blood as compared with hypothermia induced by body immersion in ice and water.

Let us now consider changes in renal and portal flow during total cardiopulmonary bypass with extracorporeal circulation both in warm and in moderately cooled animals. In normothermic animals renal and portal flows are low with low flow rates and increase with higher flow rates (Fig. 5). The same is true in the hypothermic animals although the flow rates are relatively higher as compared with control hypothermic values. This is evident when the flows are calculated in percentage of control flow values in the given temperature range (Fig. 6). For example, the renal and portal flows at a perfusion rate of 60 c.c. per kilogram per minute are approximately one-half the control values at normothermic temperatures and nearly 80 per cent at hypothermic temperatures. At normal body temperatures, the portal flow exceeds the normal with a flow rate of 80 c.c. per kilogram per minute; the renal flow is not quite normal with flow rates of 100 c.c. per kilogram per minute.

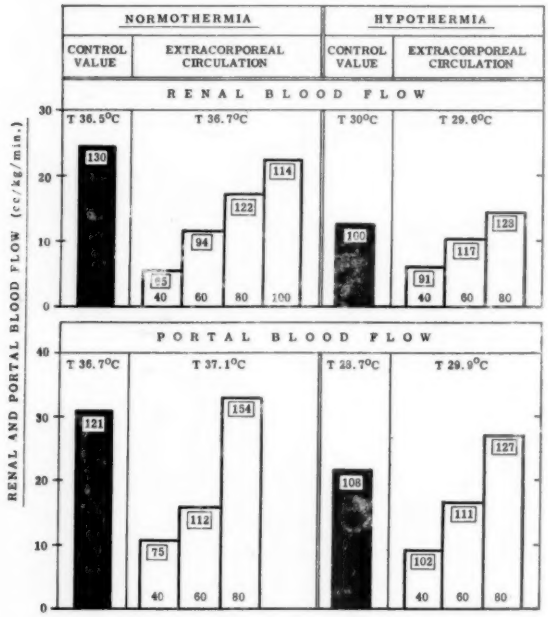


Fig. 5.—Chart comparing renal and portal blood flows in cubic centimeters per kilogram per minute during extracorporeal circulation at normothermic and hypothermic body temperatures. The figures in brackets near the top of the columns indicate average blood pressure. In this figure and in Figs. 6 and 7 the figures at the base of the columns indicate total perfusion flow rates in cubic centimeters per kilogram per minute.

With reduced body temperature both renal and portal flows are above normal hypothermic control values during perfusion at flow rates of 80 c.c. per kilogram per minute. In Fig. 7 the renal and portal flows are considered as percentage of total body circulation. Renal flows are somewhat greater with larger perfusion volumes under normothermic conditions, and under hypothermia they vary little with changing perfusion volumes. They are all in the neighborhood of the control values. The portal flows are somewhat in excess of the control values at various perfusion rates under normal body temperature conditions and somewhat less under hypothermic conditions.

Circulation through the splanchnic bed and into the liver via the portal vein constitutes a problem of surgical significance in a variety of conditions.

To mention a few, I might cite portal hypertension due to intra- or extrahepatic portal block, conditions associated with impaired liver function, and shock. We have been interested in the responses of portal venous flow to the administration of various drugs when given as a single intravenous injection.

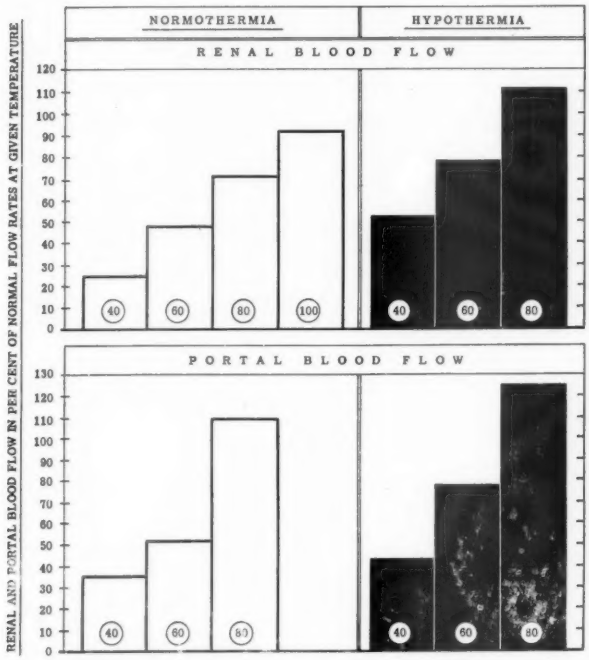


Fig. 6.—Chart showing values for renal and portal flows during extracorporeal circulation expressed as percentage of normal flows for the given body temperature range.

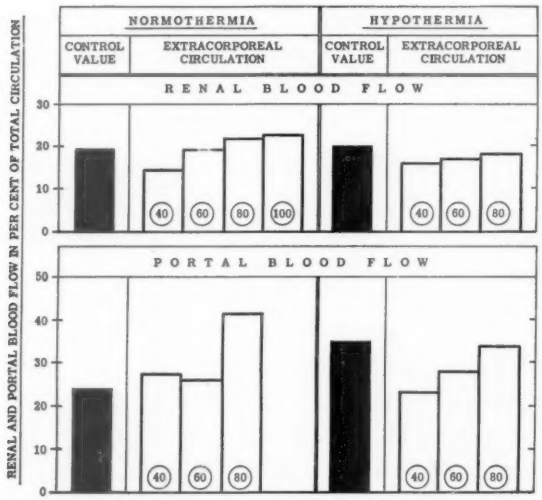


Fig. 7.—Chart showing values for renal and portal flows during extracorporeal circulation expressed as percentage of total body circulation.

Pituitrin causes a profound immediate decrease in portal flow to about 27 per cent of the control value, a decrease in portal venous oxygen saturation, and a modest rise in systemic arterial pressure. The alteration in portal flow is brief,

lasting only about 5 minutes. Pitressin causes a similar brief, and less profound, reaction. Pitocin has no appreciable effect nor does Thorazine or prostigmine. Priscoline seems to cause a very transient slight increase in portal flow lasting only about 2 minutes. Norepinephrine, Neo-Synephrine, and Aramine have roughly comparable effects. They elevate the blood pressure and cause a momentary slight fall in portal venous flow followed by a transient increase. This increase is so slight with Neo-Synephrine as to be questionable. Norepinephrine brings about an increase of about 40 per cent in portal flow, but the effect is dissipated within 5 minutes. Aramine causes a more sustained rise, one which, however, lasts only about 15 minutes. Epinephrine brings about a brief rise in portal flow without the initial transient decrease noted after administration of norepinephrine, Neo-Synephrine, and Aramine. Its effect is gone within 5 minutes. Arfonad causes a significant decrease in blood pressure and portal flow. Its effects are sometimes of brief duration but sometimes more prolonged.

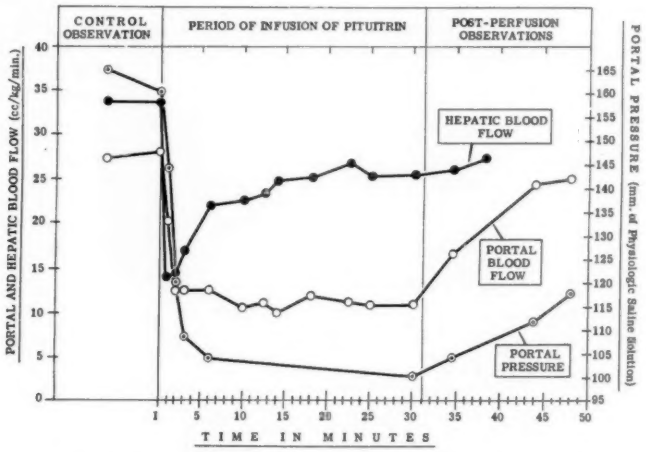


Fig. 8.—Changes in portal venous blood flow, hepatic venous flow, and portal venous pressure as a response to infusion of dilute pituitrin solution.

If Pituitrin is administered in a constant infusion to the normal dog, the portal flow can be maintained at a low level. Portal pressure is similarly kept depressed. If instead of measuring portal venous flow one measures total hepatic venous flow, one finds that the same infusion of Pituitrin causes an initial drop in flow but that after a few moments the flow rapidly rises to a much higher level but one still somewhat below the normal (Fig. 8). This difference in effect of Pituitrin upon portal and total hepatic flows is an interesting observation. If portal flow is maintained at a low level and total hepatic flow quickly rises after the initial fall, this must signify that the hepatic arterial inflow is increased. Whether this occurs as a direct response to the drug or to the mild hypertension it produces, or whether it results, on the other hand, entirely or in part from a concomitant decrease in intrahepatic resistance, I do not know.

I have previously mentioned the change in portal flow noted in general body hypothermia. Because of the recent interest of Wangensteen and his associates in local gastric cooling, portal flow has been studied under these

circumstances. The average portal flow is the same in animals subjected to local cooling of the stomach as in normal animals (Table I). Wangensteen and his group[4] have observed a marked diminution in gastric venous return during gastric cooling. If their observations, and ours, are correct one must assume that there occurs simultaneously an increase in flow to other portions of the splanchnic vascular bed. Since we find no change in the oxygen saturation of the portal venous blood, we have no lead regarding closure or opening of intragastric vascular shunts. If such changes occur in the stomach there must be other compensatory changes since the oxygen saturation of the portal blood remains the same. The fact that the portal venous circulation is not reduced by gastric cooling may be of some significance in the use of this method of treatment, particularly in cases with hepatic damage.

TABLE I. INFLUENCE OF LOCAL GASTRIC COOLING (20° C.) ON PORTAL VENOUS BLOOD FLOW AND OXYGEN SATURATION

STATE OF DOGS	BODY TEMPERATURE	PORTAL BLOOD FLOW		OXYGEN SATURATION (PER CENT)
		C.C./KG./MIN.	PER CENT OF CARDIAC OUTPUT	
Normal	36.3° C.	30.1	24.7	89.8
Local gastric cooling	34.8° C.	30.4	29.3	88.8

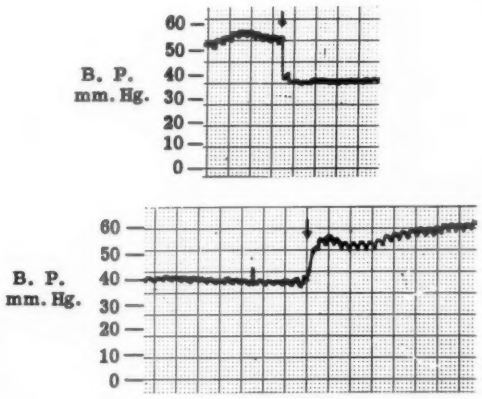


Fig. 9.—Photographs of tracings of femoral blood pressure showing (*above*) a prompt drop upon shifting an occluding aortic clamp from the infra- to the suprarenal level, and (*below*) a rise when the clamp is changed from the supra- to the infrarenal level.

Now let us consider some of the challenging and puzzling characteristics of renal blood flow. What is the mechanism which permits two small organs supplied by arteries only a few millimeters in diameter to siphon from the general circulation approximately one-fifth of the total cardiac output? Obviously such a large flow through the kidneys is possible only by virtue of extraordinarily low vascular resistance. If one measures the femoral arterial pressure and places a clamp across the aorta just caudad to the renal arteries, the pressure stabilizes around 50 to 60 mm. Hg. If one then places a clamp cephalad to the renal arteries and simultaneously removes the distal clamp, the pressure in the femoral artery falls immediately to a range of approximately 25 to 50 mm. Hg (Fig. 9). Upon reversal of the procedure a corresponding rise in femoral pressure is observed as the clamp is shifted from the supra- to the infrarenal aorta.

The fact that the vascular resistance of the kidneys is so low as to permit them to drain off a significant amount of the collateral blood flow about the clamped aorta suggests that their vascular pattern functionally resembles that of an arteriovenous fistula.

Much has been written concerning "autoregulation" of blood flow through the kidneys. Most of those who have studied this problem have found evidence to suggest that in the normal or slightly hypotensive range the kidneys do have the power of autoregulation. Some, however, have found no evidence to support this concept. We have studied the problem by regulating pressure in the renal arteries by partially occluding the aorta proximally. Under these circumstances the renal blood flow falls slightly until the arterial pressure reaches 80 mm. Hg, at which level it is approximately 85 per cent of the control value. It falls a little more rapidly as the pressure is further reduced and very rapidly below 60 mm. Hg. If one carries out the same study in dogs receiving a fairly rapid infusion of 5 per cent dextrose, the renal blood flows are quite comparable except that the progressive reduction at pressures below 70 mm. Hg is somewhat less precipitous (Fig. 10).

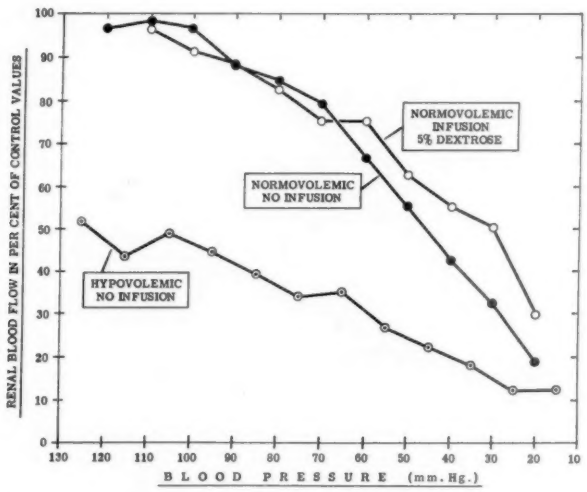


Fig. 10.—Chart showing changes in renal blood flow, expressed as percentage of control values, as the blood pressure supplying the kidneys is decreased. The pressure in the normovolemic dogs was regulated by partial occlusion of the aorta above the level of the renal arteries. The graph of the changes in hypovolemic dogs was constructed from average renal blood flow values at various systemic blood pressure levels in dogs from which 20 per cent of the blood volume had been acutely removed.

By studying renal blood flow in moderately hypovolemic dogs with varying pressures consequent to removal of 20 per cent of their blood volume, one can construct a graft in similar fashion. Although the curve of the falling renal blood flow is somewhat similar in slope to the other two, an important difference is noted. The renal blood flow values are all substantially less. For example, at a blood pressure of 90 mm. Hg, the renal blood flow in the normovolemic dog receiving no infusion is 88 per cent of the control value and the same in the normovolemic dog receiving 5 per cent dextrose solution. In contrast, the renal blood flow in the hypovolemic dog is only about 44 per cent of the control value when the blood pressure is between 90 and 100 mm. Hg. At a blood pressure

of 70 mm. Hg the normovolemic dog has a renal blood flow which is 79 per cent of normal, while the hypovolemic dog has a flow of only 35 per cent of normal at pressures ranging between 70 and 80. Because of the reported autoregulation of blood flow to the kidneys in normovolemic animals, some have analogously assumed that in the hypovolemic state a blood pressure of 80 or more signifies adequate renal blood flow. These studies suggest that this is not true, at least in the dog.

During the 100 years since Claude Bernard observed that sectioning the splanchnic nerves of an anesthetized animal increased urine flow, much has been written about the possible effects of denervation or of renal stimulation upon renal flow and function. The general impression held from the work published is that renal denervation has no demonstrable effect in the unanesthetized normovolemic animal or man and that variable results are obtained in anesthetized animals. Our own measurements of renal blood flow in the anesthetized dog 2 to 3 weeks after a unilateral splanchnicectomy and thoracolumbar sympathectomy demonstrate that the denervated kidney has an average flow about 22 per cent greater than the normally innervated kidney (Figs. 11 and 12). When shock is induced by rapid removal of blood, the flow through the denervated kidney is almost twice that of the normally innervated one. It is clear that in the dog, under the stress of anesthesia and more particularly of acutely induced hypovolemic hypotension, the denervated kidney has a greater flow than the normal kidney.

In order to test whether this observation may be of importance in recovery of the acutely damaged kidney we have carried out other experiments. We have found that exclusion of blood flow to the kidneys by aortic and bilateral renal artery occlusion for 2½ hours produces tubular necrosis and causes a mortality of 83 per cent. When the same occlusion is applied to animals previously subjected to bilateral thoracolumbar sympathectomy and splanchnicectomy, the mortality is much less. Up to the present time it is, in our hands, 50 per cent. Since some of those animals dying shortly after operation may succumb from causes related to the operation per se rather than to resultant tubular damage, the data are considered in Fig. 13 with respect to mortality in those animals which did not die within the first 3 days. The mortality in the control group is approximately 90 per cent, while it is only 30 per cent in the renally denervated group. If these data are significant, as they appear to be, one might postulate that the improved survival rate after renal circulatory arrest in animals with denervated kidneys results from better renal blood flow in the post-occlusion period.

We have carried out some studies in an effort to learn something about the minimal renal perfusion rate necessary to protect the kidneys from disastrous tubular necrosis. Direct perfusion of the renal arteries with arterialized blood at the rate of 3 c.c. per kilogram per minute for 2½ hours is followed by a high mortality; most of the animals excrete no urine during this period and the oxygen saturation of the renal venous blood falls precipitously. A perfusion rate of 5 c.c. per kilogram per minute results in a survival rate of 80 per cent. Urinary excretion during this period is absent or minimal and there is a modest drop in renal venous oxygen saturation. A perfusion rate of 7 c.c. per kilogram

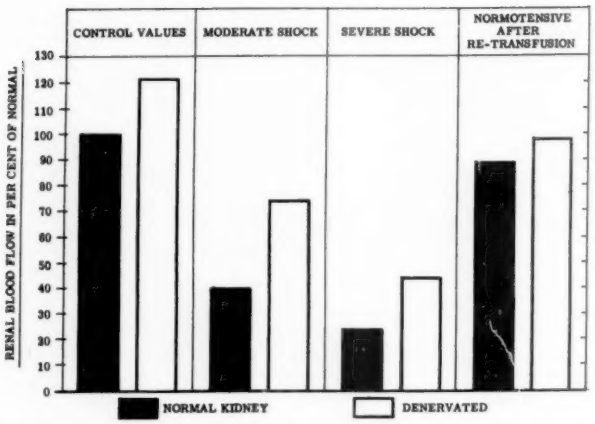


Fig. 11.—Chart comparing renal blood flows, as percentage of the normal, through the normal and the denervated kidney under normovolemic conditions and after hypotension had been induced by bleeding.

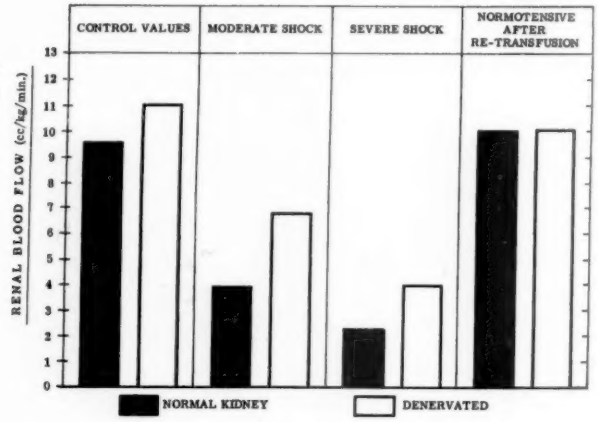


Fig. 12.—Chart comparing renal blood flows in cubic centimeters per kilogram per minute through the normally innervated and denervated kidney under normovolemic, normotensive conditions and during shock induced by acutely bleeding the animal.

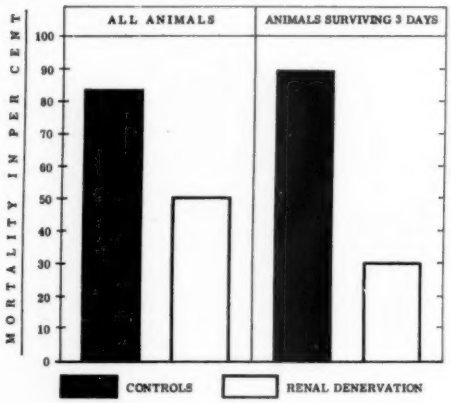


Fig. 13.—Chart showing mortality after 2½ hours of exclusion of renal arterial inflow in normal dogs and in those previously subjected to bilateral splanchnicectomy and thoracolumbar sympathectomy.

per minute is not followed by tubular damage. Urine is excreted during the period and oxygen saturation of renal venous blood is well maintained. It would appear that the dog's kidney is not damaged when renal blood flow is reduced to approximately 30 per cent of normal for a 2½ hour period.

We have been interested in the effect upon renal blood flow of certain commonly administered intravenous solutions and have made some interesting and perhaps surprising observations. If one gives intravenously to a normovolemic dog 5 per cent dextrose in water, 10 per cent dextrose in water, and physiologic saline solution, all at a fairly rapid rate (0.7 c.c. per kilogram per minute), different responses in renal blood flow are noted (Fig. 14). The saline solution does not alter the renal blood flow; the glucose solutions bring about a substantial increase. Ten per cent dextrose increases renal blood flow by approximately 75 per cent, while 5 per cent dextrose increases the flow on an average about 35 per cent. The increased flow is sustained for some time following infusion of 10 per cent dextrose but returns quickly to control levels following the administration of 5 per cent dextrose. Since the volume of water and rate of infusion were the same in these studies, we further inquired into the possible role of the glycemic blood level upon renal blood flow.

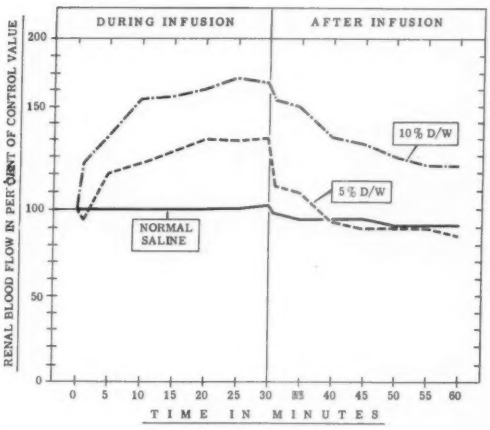


Fig. 14.

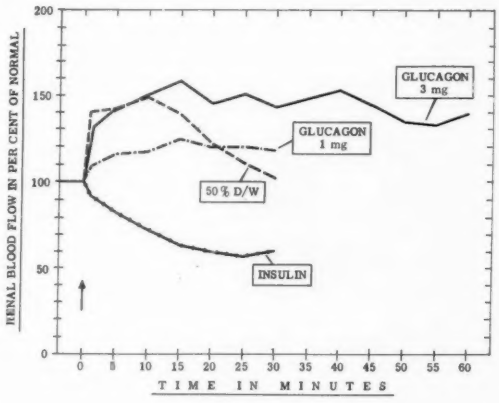


Fig. 15.

Fig. 14.—Chart showing changes in renal blood flow in the normovolemic dog upon administration of infusions of 0.9 per cent saline, 5 per cent dextrose, and 10 per cent dextrose solutions.

Fig. 15.—Chart showing changes in renal blood flow in the normovolemic dog upon administration of glucagon, 50 per cent dextrose, and insulin.

We have found that the rapid injection of 2.1 Gm. of dextrose per kilogram as 50 per cent dextrose in water (37.5 to 53 c.c.) also causes an increase in renal blood flow, almost to 50 per cent above control levels. Small (1 mg.) and larger (3 mg.) doses of glucagon similarly increase renal blood flow, while insulin decreases it (Fig. 15). That there is some correlation between the level of blood sugar and renal blood flow is evident (Fig. 16). On the other hand, one must consider the possibility that intravenous dextrose solutions may bring about a greater renal blood flow by increasing the blood volume or cardiac output. The same amount of intravenously administered 50 per cent dextrose in water used in these studies does indeed induce an immediate increase in cardiac

output which rises to a level approximately twice the control value. This marked increase is momentary. The cardiac output falls precipitously to a level which 4 minutes later is roughly 40 per cent above normal and then slowly falls further throughout the remaining 25 minutes during which the study was carried out. Glucagon, which produces a similar rise in renal blood flow, does not, however, increase cardiac output. Two minutes after the injection of 50 per cent dextrose a rise in blood volume to approximately 40 per cent above the normal level is noted; 5 minutes later it is normal. Glucagon brings about no increase in blood volume, or at best a trivial increase. Certainly the administration of 250 c.c. of whole blood increases blood volume more and causes no increase in renal blood flow in the normal dog. Furthermore, insulin, which decreases renal blood flow, does not bring about a reduction in blood volume. We must conclude, therefore, that the data at hand suggest that the effects noted upon renal blood flow from intravenously administered dextrose, glucagon, and insulin are related principally to changes in blood sugar levels.

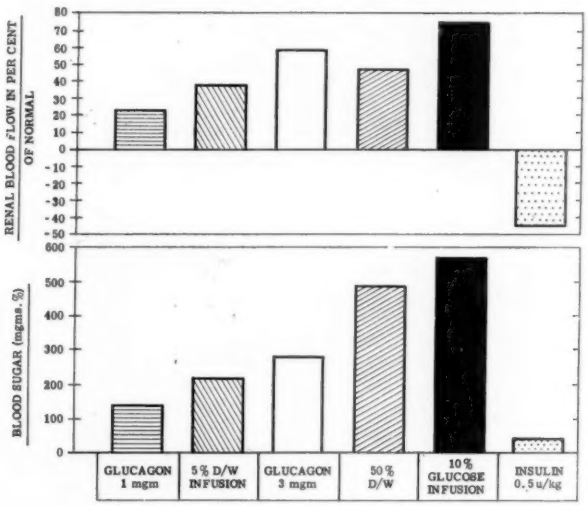


Fig. 16.—Chart showing relationship between level of blood sugar and renal blood flow.

Hypertonic saline (5 per cent) increases renal blood flow, though not to the extent of 10 per cent dextrose. It produces a greater though more delayed diuresis.

Interestingly enough, the oxygen saturation of renal venous blood is about the same at the end of an infusion of normal saline solution and after injection of a small dose of glucagon, appreciably increased after infusion of 5 or 10 per cent dextrose and after administration of a larger amount of glucagon, somewhat reduced below the control level after injection of 50 per cent dextrose and even more so at the end of an infusion of hypertonic saline solution. These observations would lead one to conjecture that certain solutions increase renal blood flow proportionately more than they increase the work load of the kidney, while others increase the renal metabolic requirements proportionately more than they increase blood flow. Insulin produces not only a reduction in flow but also a decrease in renal venous oxygen saturation.

The moderately hypovolemic dog, following removal of 20 per cent of his blood volume, undergoes a similar, dramatic increase in renal blood flow as a response to the infusion of 10 per cent dextrose, 50 per cent dextrose,* and to glucagon (Fig. 17). The reinfusion of blood itself brings about an immediate return of renal blood flow to control levels. Physiologic saline, which in the normovolemic dog causes no change in renal blood flow, increases flow in the hypovolemic dog. The most pronounced changes in arterial blood pressure after such infusions are seen on the administration of whole blood and 50 per cent dextrose. The former causes a prompt rise to slightly above control levels with a gradual fall to about 90 per cent of the control value. The latter is associated with an almost immediate fall in blood pressure of extremely short duration followed by a rise to slightly higher levels than those which existed at the time of the injection.

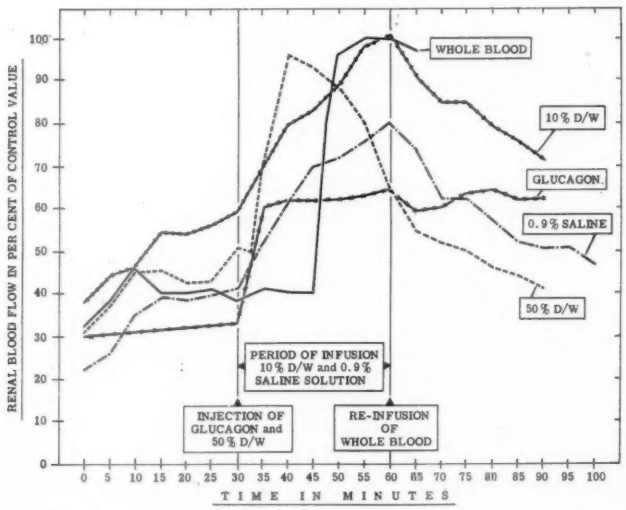


Fig. 17.—Chart showing changes in renal blood flow consequent to infusions of normal saline and 10 per cent dextrose, to the administration of glucagon and 50 per cent dextrose, and to blood replacement in the moderately hypovolemic dog (removal of 20 per cent of the blood volume). During the control period after bleeding, the renal blood flow tends to increase a bit but then levels off much as is seen here in the more prolonged control period before re-infusion of whole blood.

If these observations in the dog are relevant in the human, they might well be taken into consideration in managing the patient with impaired renal function.

Finally, I should like to mention certain observations relating to changes in renal blood flow consequent to the intravenous administration of certain pharmacologic agents in common surgical usage. A single injection of Adrenaline causes a rise in blood pressure to about 50 per cent above normal and a concomitant decrease in renal flow to about 27 per cent below control levels. Both effects are transient and are dissipated within 5 minutes. Norepinephrine causes a similar brief, but more pronounced, rise in blood pressure and fall in renal blood flow. Aramine produces a more pronounced and sustained rise in

*Half of the amount of 50 per cent dextrose used in the other experiments was given. In this study the 10 per cent dextrose and normal saline were infused at a rate so that during a 30-minute period an amount of fluid equal in volume to that of the blood that had been withdrawn was given.

blood pressure and fall in renal blood flow. Priscoline brings about a slight-to-moderate rise in renal blood flow, and chloropromazine and Pituitrin a questionable slight decrease in renal flow.

With a constant infusion of Adrenaline two types of responses are observed. In some animals there is a slight rise in blood pressure with no change in renal flow on the administration of a dilute solution, while with more concentrated solutions a more pronounced rise in blood pressure develops along with a slight decrease in renal flow. After the infusion the blood pressure in the latter animals drops to below control values and the renal flow rises to levels slightly above normal. In other animals a dilute solution causes a very slight rise in blood pressure and a slight rise, or no change, in renal blood flow, while more concentrated solutions bring about a slight increase in blood pressure and a marked increase in renal blood flow. In the latter animals the blood pressure and renal blood flow values quickly return to normal after the infusion ceases.

An infusion of very dilute norepinephrine brings about a slight rise in blood pressure and renal blood flow. A more concentrated solution, however, brings about a significant rise in blood pressure and a very significant fall in renal flow to values about 50 per cent below normal. Dilute solutions of Aramine also cause a slight rise in blood pressure and renal blood flow, while more concentrated solutions again bring about a rise in blood pressure and a pronounced fall in renal flow to approximately 50 per cent below normal values.

When these drugs are given as a single injection during cardiopulmonary bypass with extracorporeal circulation, a situation in which the blood volume is apparently unaltered and the total circulation per unit of time is fixed, a response similar to that observed in normal dogs is seen (Figs. 18 and 19). Although our studies are as yet incomplete, the response of moderately hypovolemic dogs also seems to be rather similar. In general, both norepinephrine and Aramine bring about a rise in blood pressure and also a slight rise in renal blood flow, provided there is only a slight or moderate rise in blood pressure. If, however, the blood pressure is elevated markedly (to 180 mm. Hg or higher), the renal blood flow decreases precipitously at the same time.

Regardless of the state of the animal and the agent employed, our studies suggest that a vasopressor which increases the dog's mean blood pressure to about 180 mm. Hg or more tends to reduce significantly renal blood flow.

DISCUSSION

I have presented certain observations relating to the distribution of blood flow with only brief comments concerning their possible significance. When they are reported in more detail the implications of these findings will be discussed more thoroughly. It is entirely likely that some, and perhaps most, of the information obtained from these animal studies may be of some significance in understanding and caring for the human patient. On the other hand, this assumption cannot be taken for granted. As is true with any animal experimentation, it is important to repeat as much of this work on human beings as can possibly be carried out with safety. It is equally evident that these studies,

which have related almost exclusively to the distribution of blood flow, should be amplified wherever possible by similar studies designed to learn more about concomitant changes in organ function as the blood flow through it is changed.

The ability of the body to make certain adjustments to changing circumstances in order to maintain a relatively constant internal environment constitutes one of the great wonders of the biologic world. This continuing miracle has prompted justifiable reverence for the "wisdom of the human body." As we begin to understand more about the alterations consequent to certain noxious

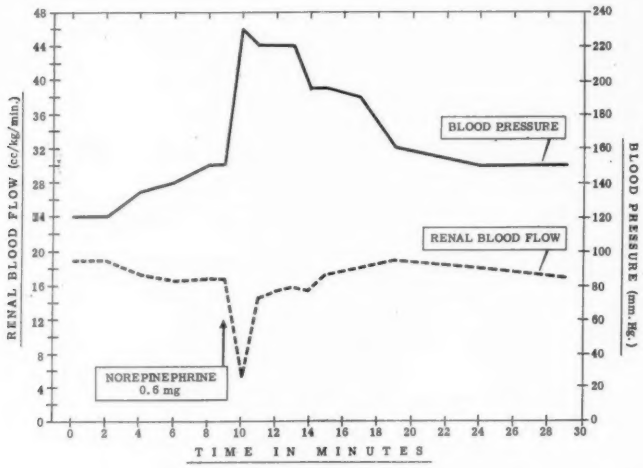


Fig. 18.—Chart showing typical response of blood pressure and renal blood flow to norepinephrine in a dog during total cardiopulmonary bypass and extracorporeal circulation (80 c.c. per kilogram per minute).

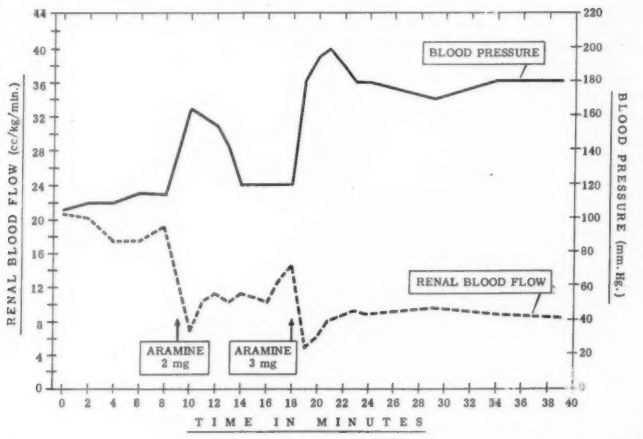


Fig. 19.—Chart showing typical responses of blood pressure and renal blood flow to injections of Aramine in a dog during total cardiopulmonary bypass with extracorporeal circulation (80 c.c. per kilogram per minute).

influences, we are, however, persuaded that the body's response is not always a wise, favorable, health-preserving one. An example is seen in the dramatic, sharp reduction in renal blood flow, often to dangerously low levels, which occurs as a consequence of even mild or moderate blood loss. Of more significance is the fact that therapeutic measures instituted primarily to bring about one

result which is considered desirable may evoke other responses which are perhaps detrimental. Once these multiple effects are well understood, we should be in a better position to improve our care of patients. Such knowledge might lead to the discovery that certain agents evoke predominantly good responses. Undoubtedly, other agents will be found to exert certain influences which are beneficial and others which are detrimental. Their usage will then be based on a firm footing with clear recognition of the concomitant adverse responses which are judged to be outweighed in importance by others known to be beneficial.

This incomplete inquiry into certain problems relating to the distribution of blood flow and factors which modify it encourages me to believe that continued efforts of this sort may be rewarding.

REFERENCES

1. DeBakey, M. E., Burch, G., Ray, T., and Ochsner, A.: The "Borrowing-Lending" Hemodynamic Phenomenon (Hemometakinesia) and Its Therapeutic Application in Peripheral Vascular Disturbances, Ann. Surg. **126**: 850, 1947.
2. Hardin, R. A., and Scott, J. B.: Cardiac Cooling in Normothermic Dogs, S. Forum. In press.
3. King, H., and Bounous, G.: Cardiac Input in Hypothermia, Surg. Gynec. & Obst. **107**: 753, 1958.
4. Wangensteen, O. H., Salmon, P. H., Griffen, W. O., Jr., Paterson, J. R. S., and Fattah, F.: Studies of Local Gastric Cooling as Related to Peptic Ulcer, Ann. Surg. **150**: 346, 1959.

PRESENTATION OF DR. LUTHER LEONIDAS HILL MEMORIAL PLAQUE

AT its annual meeting in Atlantic City on June 7, 1959, the Society for Vascular Surgery presented to Senator Lister Hill of Alabama a plaque commemorating the pioneering contributions to cardiovascular surgery of his father, Dr. Luther L. Hill. Dr. Hill was the first American doctor successfully to suture a wound of the heart, which entered one of the heart chambers.

Dr. Harris B. Shumacker, President of the Society, in the presence of Mrs. Hill and the Senator's two sisters, presented the plaque on behalf of the Society with the following remarks:

Dr. Shumacker.—Ladies and gentlemen, during the past year the Council of this Society has taken a rather unusual action and one which I know has the wholehearted approval of the membership of this Society and of our guests. It has been unusual in the annals of this organization to do anything especially to honor a single individual. We have, as you know, established a Lectureship in the name of one of our most distinguished members, Dr. John Holman. We have also honored the name of Dr. Rudolph Matas, one of the greatest contributors to the field of vascular surgery. I have before me the gavel which shortly will be turned over to the new President, and on this gavel the following is inscribed:

> May this gavel made from the oak of the Old Charity Hospital, New Orleans, perpetuate the name and honor of Rudolph Matas, pioneer vascular surgeon.

The action which we have taken has prompted us to bring here today Senator Lister Hill, and we are delighted that his wife and sisters are with him.

In 1862 in Montgomery, Alabama, was born Dr. Luther Hill, who had a long and very productive life. After receiving his medical education in New York and Philadelphia and further expanding it by experiences in Europe, he settled down into the practice of surgery in Montgomery. Because of the very significant contributions which he made in the field of cardiovascular surgery, the Society for Vascular Surgery, Senator Hill, wishes to present to you this plaque.

Among other notable contributions, Dr. Luther Hill was the first in this country successfully to repair a cardiac wound which penetrated into one of the chambers of the heart. Senator Hill, I am quite confident that your father would be even more pleased today to know that his son has maintained such a vivid interest, from the lay, political, and professional points of view, in the field of medicine and has been the guiding influence in our national Congress for constructive legislation aimed to better the health of the citizens of this country than he would be by this tribute from our Society recognizing his own work.

The plaque, Senator Hill, is, I think, a very pretty one. Upon it is inscribed the initials of the Society for Vascular Surgery and the following:

> This plaque is gratefully dedicated in appreciation of the pioneering contributions in the field of cardiovascular surgery made by Dr. L. L. Hill, Montgomery, Alabama, 1862-1946.
>
> The Society for Vascular Surgery, 1959.

Senator Hill, we should like to present to you for your keeping a copy of this plaque which has been placed in a suitable frame, as well as the plaque itself, which you may deposit wherever you wish it to be kept.

I congratulate you and your family.

Senator Hill then accepted the plaque with the following remarks:

Senator Hill.—President Shumacker, Members of the Society for Vascular Surgery, and friends, if words spoke the language of the heart I could better tell you how proud and how grateful I am for the honor you have done my father.

I know full well how profound would be his pride, how deep his gratitude for the accolade bestowed upon him by you, the members of the medical profession, the profession which was his very life, the profession to which he gave his all.

Ever did he cherish the friendship and the esteem of his fellow doctors. Let his own words tell that story. On the occasion of his fifty years in the practice, at a banquet given in his honor by his friends, the doctors of Alabama, he said,

> I care not what a man's mental endowments are nor what his professional attainments may be, unless he is true to his ideals, and has the approval, the sympathy of his fellows, his life is a failure, for the dominant and triumphant tone of life is love.

He then quoted Oliver Wendell Holmes:

> You can unfrock a clergyman, unwed a husband, but once a doctor always a doctor.

To this he added,

> Of course, this does not mean that a doctor may not take up another vocation, but that he cries out to the profession as did Holmes when he became a literary man: "Claim me, hold me, call me brother still."

It was at this banquet that that great pioneer in vascular surgery, Dr. Rudolph Matas of New Orleans, paid this tribute to my father:

> On an occasion when he is surrounded by a circle of friends . . . , who honor him for the magnificent example he has set to the younger generations, his sleepless pursuit of knowledge, his integrity of character and the purity of his purpose in maintaining the dignity and the highest ideals of his profession, it would seem superfluous to chant a hymn of his merits—merits which are known to all who have enjoyed the privilege of his friendship and by the thousands whom he has comforted and made happier by his science, his skill . . . his unfailing understanding and human sympathy. We who love him for what he is to us personally and professionally, hail the advent of his golden jubilee with the acclaim that is due to the prize winner in the race of life's noblest achievements.

At the age of nineteen, my father was graduated from New York University Medical School, the following year he was graduated from Jefferson Medical College in Philadelphia, after which he spent a year at Wyeth's New York Polyclinic School, and then studied for some months under Joseph Lister at King's College Hospital in London. In 1881, after his graduation in New York, he was admitted to the practice of medicine in Alabama. In that very year John B. Roberts of Philadelphia predicted that the human heart would be sutured, and Billroth, the great Viennese surgeon, replied, "Any man that attempts it will lose the respect of the profession."

Though my father had never seen an abdominal operation until he performed one himself, and though he was conversant with Billroth's statement, he went to work to study wounds of the heart and their possible suture. In 1900 he wrote an article, published in *The Medical Record*, on "Wounds of the Heart, With a Report of Seventeen Cases of Heart Suture."

In 1902 came the opportunity for which he had been eagerly waiting. On September 14 of that year he successfully sutured the heart of a 13-year-old Negro boy where the cavity of the heart had been penetrated by the blade of a knife. May I remind you, my friends, that this, the first successful suture in these United States, was performed by the flickering light of two kerosene lamps on an old kitchen table in a Negro shack. In *The Medical Record* of Nov. 29, 1902, my father reported the case.

If my father were here today I know he would congratulate you on the wonderful progress you have made in vascular surgery. I know he would rejoice over the magnificent work that you are doing today. I know he would remind us that the doctor is ever the central figure in the drama of medical care. He would emphasize that all else is to assist the doctor to do his best, to help him achieve the best possible results. He would warn us that if the doctor is to do his best he must remain free and uncontrolled; that only as the doctor finds the inspiration and enjoys the right of individual action that freedom gives—to explore, to inquire, to discover, to serve in his own way—only then can he give his best, only then can we continue the marvelous progress of American medicine. With profound thanks to you, may I say that in this world of confusion, of uncertainty and of danger, we must preserve the incentives of our free medical system.

In words that live today, yet spoken in 1899 on the occasion of the fiftieth anniversary of the Medical Society of Philadelphia County, Dr. J. Chalmers da Costa of Philadelphia declared:

> Our fathers did wonders with the resources they could command. The lesson of their lives is largely one of dignity, self-sacrifice, devotion to science, and regard for the bonds of professional conduct and duty, and carelessness as to wealth and fame.

With this heritage and in the spirit of freedom, we shall and must go forward together into the ever-widening frontiers of medical discovery and medical knowledge. In the confidence of knowledge, in the strength of integrity,

in the tenderness of sympathy, in the fellowship of humility, and in the love of God, we shall and must continue to wage together the never-ending battle for the health of our people, for the health of all peoples.

Following the acceptance of the plaque, the Society presented Senator Hill with a scroll in honor of and appreciation for his own contributions toward the advancement of medical research and public health. Senator Hill's efforts in this field at the state and national levels are well known. He became the first senator ever to receive the special Lasker Award for public service to the nation's health when the Award was formally presented to him at the Eighty-seventh Annual Meeting of the American Public Health Association. Only four of the special Awards, one of the highest honors in the field of public health, have been presented in the past, all to doctors. Senator Hill was selected for the last Award by 15 physicians appointed by the American Health Association. The Alabama senator is chairman of the Senate committees handling both health legislation and health appropriations.

The text of the scroll presented by the Society for Vascular Surgery to Senator Hill is as follows:

> In appreciation of the outstanding contributions to the health of our people made by Senator Lister Hill and in recognition of his untiring efforts and achievements in advancing cardiovascular research, this citation is gratefully presented.
>
> The Society for Vascular Surgery,
> June 7, 1959.

PERSISTENT EXPERIMENTAL AORTIC ANEURYSMS IN DOGS

STEVEN G. ECONOMOU, M.D., C. BRUCE TAYLOR, M.D.,
EDWARD J. BEATTIE, JR., M.D., AND CARL B. DAVIS, JR., M.D., CHICAGO, ILL.
(From the Departments of Surgery and Pathology, Presbyterian–St. Luke's Hospital)

THIS investigation of the effect of intramural injection of contrast material, as might occur with faulty aortography, was prompted by the increasing number and variety of complications secondary to this procedure.[1-3]

EXPERIMENT I

Methods.—Twenty mongrel dogs of average size were used. The most commonly used aortographic contrast material, 70 per cent acetrizoate (Urokon), was chosen for our study. Initially the abdominal aorta was utilized, but paraplegia as a result of accidental intraluminal escape of the contrast material caused us to shift to the thoracic aorta. With the animal under general endotracheal anesthesia, the aorta was exposed through a left thoracotomy. The parietal pleura was dissected from the proposed area of injection. Acetrizoate (70 per cent concentration), in amounts proportional to that used in human beings (1 to 2 c.c.), was injected intramurally with a No. 27-gauge needle. The needle was bent to a 45 degree curve so that the orifice of the needle could be maintained completely within the vessel wall with more certainty during the injection (Fig. 1). A fair amount of pressure was necessary to perform the injection and the contrast material could be seen to dissect laterally through the mid-portion of the media for about 1 cm. around the needle puncture site.

An effort was made to study the effect of 70 per cent acetrizoate on atherosclerotic vessels in rabbits fed a commercial diet containing 1 per cent cholesterol and 5 per cent corn oil. The very thin-walled arteries made intramural injection extremely difficult. Also, frequent accidental intraluminal injection of acetrizoate produced paraplegia and early death in the animals. Accordingly, this avenue of investigation was abandoned.

Results.—The dogs were sacrificed at intervals beginning the first week and extending to the thirtieth week. There were occasional adhesions of lung to the thoracotomy wound and always dense adhesions between lung and the injected site of the aorta. Gross aneurysms were observed as early as the third week and as late as the thirtieth week which was the maximum time the animals were kept. This observation was significant not only in demonstrating the potential

Supported by the Chicago, the Illinois, and the American Heart Associations, the Life Insurance Medical Research Fund, the Ames Company of Elkhart, Indiana, the National Heart Institute, National Institutes of Health (H-1630, H-3215), and the Otho S. A. Sprague Memorial Institute.

Read at the Thirteenth Annual Meeting of the Society for Vascular Surgery, Atlantic City, N. J., June 7, 1959.

and actual damage possible by intramurally placed 70 per cent acetrizoate, but because it is one of the few ways whereby low grade but sustained damage to a vessel wall can be experimentally produced.

The aortic aneurysms were fixed in 10 per cent formalin. Blocks for microscopic sections were cut; these included the complete diameters of the aneurysms and normal aortic tissue at both margins. Microscopic sections were stained with hematoxylin and eosin, with Weigert's elastic tissue stain, and with Mallory's trichrome stain.

Microscopically, the lesions demonstrated a rather low grade, or smoldering, progressive destruction of the mid-portion of the media. Earlier lesions (1 to 2 weeks old) showed hemorrhage between elastic lamellae with early evidences of tissue necrosis (Fig. 2). Later lesions (3 to 6 weeks old) showed continuing resorption of the intramural hemorrhage with much more striking destruction of elastic lamellae, collagen, and cellular elements in the mid-portion of the media. Late lesions (as old as 30 weeks) showed complete loss of structural elements in the site of medial injection. The total thickness of the aortic wall at this site was markedly reduced to the point of translucency in many cases.

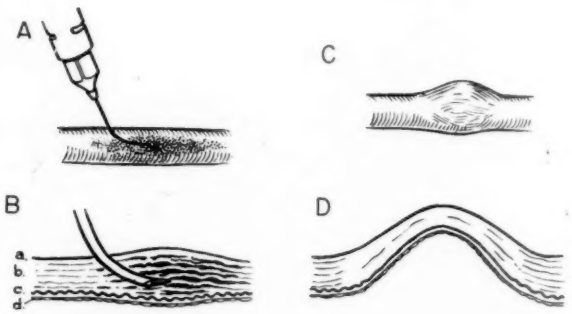


Fig. 1.—Diagrammatic illustration of method of intramural injection of 70 per cent acetrizoate. *A*, A 45 degree curve in the needle makes it easier to maintain the orifice of the needle in an intramural position. *B*, The contrast material dissects laterally and separates the elastic lamellae. *a*, Adventitia; *b*, media; *c*, internal elastic membrane; *d*, endothelium. *C*, Aneurysmal dilatation observed as early as 3 weeks after intramural injection of the contrast material. *D*, Microscopically there is destruction of the central portion of the media. There is no apparent injury to the internal elastic membrane and subendothelial layer. No subendothelial reparative scar was noted as long as 30 weeks after intramural injection of acetrizoate.

Significantly, there was no evidence of repair of this damage even in aortas injured as long as 30 weeks previously. This was in sharp contrast to earlier work by Taylor and associates.[4, 5] Using a "cooling gun" which worked on the principle of rapidly expanding CO_2, they quick-froze for one minute the full thickness of abdominal aortas (Fig. 3). The destruction of the full thickness of the aortic wall resulted in the immediate formation of aneurysms. Within 3 weeks marked subendothelial proliferation had occurred. These multipotential cells had already begun to form elastic tissue and collagen fibers. Six weeks after injury these subendothelial cells had developed into an essentially new segment of arterial wall with abundant elastic tissue and smooth muscle cells. These well-formed arterial scars had restored sufficient new functional arterial tissue to repair the aneurysm present 6 weeks earlier. It appeared that stimulation by injury of the multipotential subendothelial layer was the trigger

mechanism to a reparative process. This was supported by the observation that removal of only the intima and internal elastic membrane (Fig. 4) stimulated profuse subendothelial proliferation with rapid repair of the intimal defect.[6]

EXPERIMENT II

Methods.—To further test the thesis that stimulation of the multipotent subendothelial layer is a prerequisite to arterial repair, we produced aortic aneurysms in 10 animals by removal of the outer 60 to 70 per cent of the media while leaving the remaining media and the internal elastic membrane and subendothelial layer intact. The technique was as follows. With the animal under general endothracheal anesthesia, the aorta was exposed through a left thoracotomy.

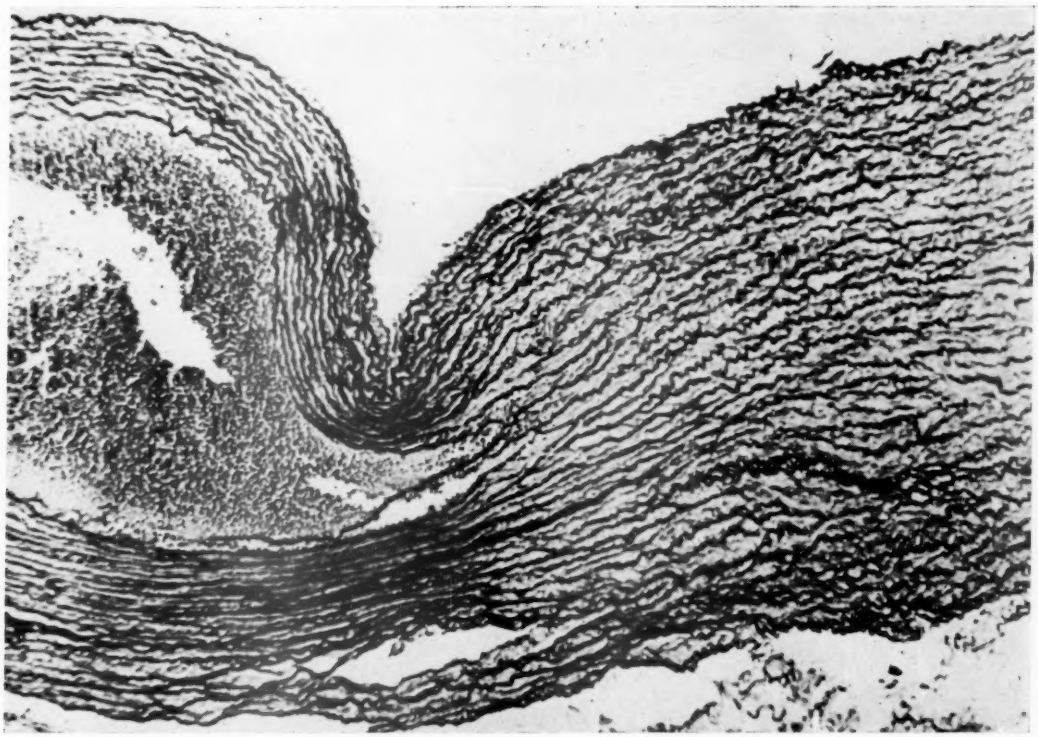


Fig. 2.—Photomicrograph of Weigert's elastic tissue stain of half of lesion 1 week after injection of 1 c.c. of 70 per cent acetrizoate intramurally. There is a large hematoma at this time separating elastic lamellae on the left. Some elastic lamellae are fragmented and others are straightened due to death of smooth muscle cells and other cellular elements.

The adventitia was dissected away. With gentle strokes of the scalpel, the media was cut through in layers. By not occluding the circulation during the dissection, the pressure within the vessel produced a bulge at the dissected area which acted as a guide to the depth of cutting necessary. Once the desired depth of dissection was reached, the media could be peeled along a cleavage plane at the depth of the dissection. An ellipse of media was then cut away, allowing full development of the aneurysm (Figs. 5 and 6). Optimal aneurysmal formation occurred when approximately 70 per cent of the media was

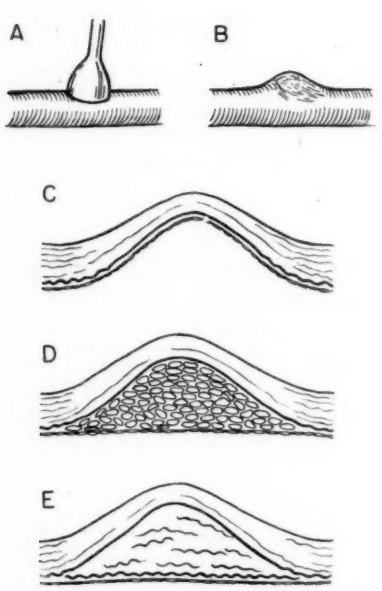


Fig. 3.

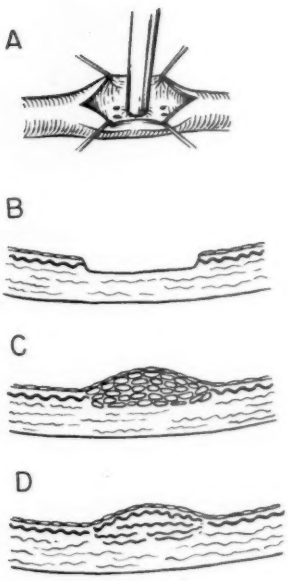


Fig. 4.

Fig. 3.—Diagrammatic illustrations of effect of transmural freezing (*A*) on arterial tissue. Immediately after freezing aneurysm develops at site of freezing (*B*) and persists for several weeks. *C* illustrates straightening and disruption of elastic lamellae and dissolution of killed cells in area frozen. During the first 2 weeks there was no proliferation of multipotential subendothelial cells (*C*); however, during the third week these cells proliferated abundantly and essentially filled the aneurysmal defect (*D*). The multipotential cells differentiated and formed an essentially new vascular wall at about the sixth week (*E*). After 6 weeks mature cells and new elastic and collagen fibers have restored the artery to its original caliber and repaired the aneurysm.

Fig. 4.—Diagrammatic illustration of response of aorta to stripping of endothelium and subjacent internal elastic lamella. A shallow circular cut is made by gently rotating a ½ inch diameter cork borer (*A*). Endothelium, subendothelium, and internal elastic membrane are then dissected away leaving shallow circular defect (*B*). As shown in *C* at about 3 weeks a thick tuft of subendothelial cells and a thin layer of endothelial cells cover the defect. At about 6 weeks the intimal scar covering the small area of injury has differentiated into mature vascular scar tissue with elastic and collagen fibers (*D*).

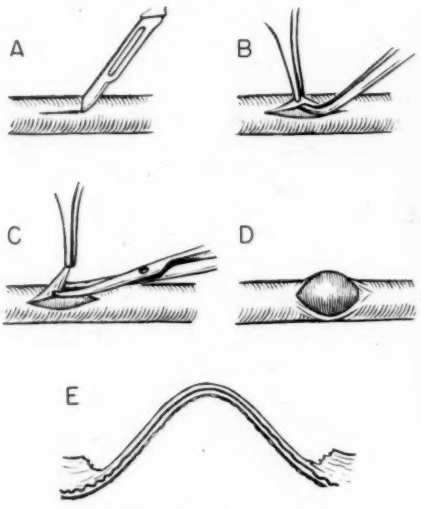


Fig. 5.

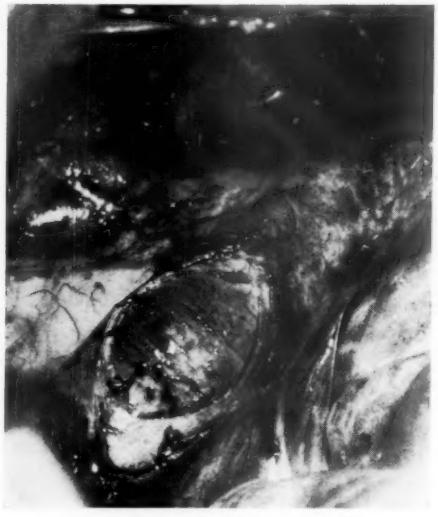


Fig. 6.

Fig. 5.—Diagrammatic presentation of formation of aneurysm by stripping of outer 70 per cent of the media. *A*, With gentle strokes of the scalpel the desired depth of dissection is reached. *B* and *C*, at the depth of the incision the media could be peeled along a cleavage plane. An ellipse of the dissected media is excised, allowing full development of the aneurysm (*D*). With removal of more than 70 per cent of the media, acute rupture of the resultant aneurysm occurred. *E*, Aneurysmal formation showing stretching of internal elastic membrane. Even though 70 per cent of the media had been removed without injury to the subendothelial space and internal elastic membrane, there was no reparative response.

Fig. 6.—Photograph of aneurysm of thoracic aorta produced by surgical removal of outer 70 per cent of media.

excised. A lesser dissection would not produce an aneurysm whereas removal of more than 70 per cent of the media resulted in acute rupture of the resultant aneurysm.

Results.—The animals were sacrificed from the first to the thirtieth week. All the aneurysms had persisted. Incidentally, production of hypertension before and after the production of aneurysms did not result in larger aneurysms at the time the animals were sacrificed. As expected, on microscopic examination no attempt at repair of the aneurysms was evident as long as 30 weeks later (Fig. 5, *E*).

EXPERIMENT III

Methods.—An experiment was designed to demonstrate within a single aneurysm the necessity of subendothelial stimulation if a reparative process were to take place. Aneurysms were created by medial stripping, and one-half of the aneurysm was quick-frozen in its entire thickness for 2 minutes. The other one-half of the aneurysm was not disturbed any further (Fig. 7).

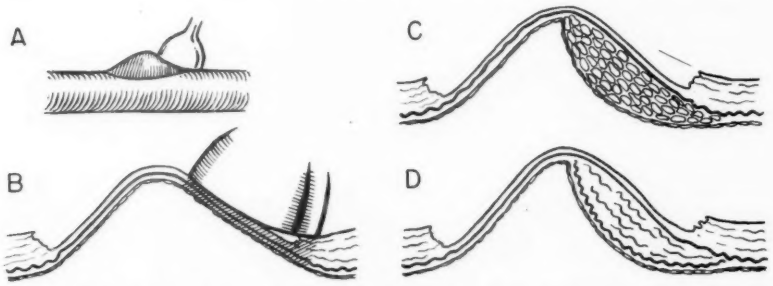


Fig. 7.—*A* and *B*, Transmural freezing of one-half of aneurysm which had been produced by excision of 70 per cent of outer media. The other half was left undisturbed. *C*, Whereas there was no reparative process in the portion of the aneurysm which was left undisturbed, there was brisk proliferation of subendothelial cells in the portion of the aneurysm that had been transmurally frozen. *D*, The multipotential subendothelial cells have differentiated and formed an essentially new arterial wall in a thickened intima at the site of transmural freezing.

Fig. 8.—Photomicrograph of Weigert's elastic tissue stain of lesion produced by excision of outer media. Cut ends of outer elastic lamellae are visible on the lower surface of the aorta near each lateral margin of the photograph. After excision of the outer media two-thirds of the aorta on the right was transmurally frozen; 2 months later this right-hand portion of the aneurysm showed a thick intimal scar containing a rich elastic network. The aneurysmal dilatation on this side of the aneurysm was markedly corrected by this thick intimal scar.

Results.—The aneurysms from these animals showed the development of a thick subendothelial scar over the half of the aneurysm that had been transmurally quick-frozen. Marked correction of the aneurysmal dilatation of the half of the aneurysm with this subendothelial scar was apparent both grossly and microscopically (Fig. 8).

DISCUSSION

Repair of injured or degenerated arterial tissue is a somewhat unique process. The mature cells in the media make essentially no contribution to the repair process. Following injury, scattered, primitive mesenchymal cells lying in the subendothelial space proliferate rapidly. Subsequently these cells differentiate and participate in the formation of an intimal arterial scar containing all elements found in the medial coats of arteries. The scar consists of an essentially new arterial wall lying on the intimal surface of the damaged media.[4, 5]

The new findings reported here have demonstrated that medial damage alone does not initiate the typical arterial repair mechanisms. The aneurysmal dilatation and stretching of the weakened arterial wall do not seem to stimulate the multipotent mesenchymal cells to proliferate and form new vascular tissue. The findings reported do demonstrate that the typical arterial repair response is elicited when the arterial injury includes damage to the subendothelial space and the internal elastic membrane.

SUMMARY AND CONCLUSIONS

1. Persistent experimental aortic aneurysms have been produced in the aortas of dogs by the local injection of 1 to 2 c.c. of 70 per cent acetrizoate into the mid-portion of the media of each animal.

2. Aortic aneurysms also developed and persisted after surgical resection of 60 to 70 per cent of the outer media.

3. These aneurysms persisted, unchanged, for 30 weeks, the longest period studied. They failed to show the usual intimal scar observed following transmural freezing injury of arterial walls.

4. Transmural freezing of one-half of an aneurysm made by surgical resection of the outer media resulted in marked correction of this portion of the aneurysm by a thick intimal scar.

5. These studies indicate that loss of medial structure alone does not elicit a typical arterial repair response. In order for an intimal arterial repair response to occur, the subendothelial tissue and internal elastic membrane must be stimulated by injury.

REFERENCES

1. Boyarsky, S.: Paraplegia Following Translumbar Aortography, J. A. M. A. **156**: 588-602, 1954.
2. Deterling, Jr., A. R.: Direct Retrograde Aortography, SURGERY **31**: 88-114, 1952.
3. McAffe, J. G., and Wilson, J. K. V.: A Review of the Complications of Translumbar Aortography, Am. J. Roentgenol. **75**: 956-970, 1956.
4. Taylor, C. B.: The Reaction of Arteries to Injury by Physical Agents With a Discussion of Arterial Repair and Its Relationship to Atherosclerosis, *in* Symposium on Atherosclerosis, Publication 338 of National Academy of Sciences, National Research Council, 1955.
5. Taylor, C. B., Baldwin, D., and Hass, G. M.: Localized Arteriosclerotic Lesions Induced in the Aorta of the Juvenile Rabbit by Freezing, Arch. Path. **49**: 623-640, 1950.
6. Taylor, C. B., Cox, G. E., Stumpe, M. A., and Weinberg, M.: Endothelial Regeneration, Circulation **14**: 487, 1956.

Discussion

DR. ORMAND C. JULIAN (Chicago, Ill.).—It is fortunate that the accidents which are implied by the experimental work which might occur in clinical aortography or clinical arteriography are apparently rare.

The thesis of the paper is extremely well presented. I would, however, like to present an alternate possibility to the one expressed by the authors as the manner of stimulation of the subendothelial areas for repair.

It seems to me inherent in the experiments presented this morning and also those of Taylor is the presence or absence of damage to the endothelium. The diagrams show very clearly that the presence of endothelial damage allows the deposition of a mural clot, blood cells bringing with them other potential cells which then go on to reconstitute the vessel wall.

I would suggest that we still have the possibility that the repair comes from blood stream cell rather than from the cells of the subendothelium which fits in rather well with general ideas in repair of injury.

This is seen sometimes in clinical cases of traumatic aneurysms of the thoracic aorta, which are frequently devoid of mural clots, remaining thin-walled aneurysms and persisting for long periods of time. These aneurysms failed to develop clots within them because the endothelium repaired too rapidly for external deposition of clot material, and therefore, the reconstruction of the vascular wall with obliteration of the aneurysm does not occur.

DR. RALPH A. DETERLING, JR. (New York, N. Y.).—In reviewing several hundred animals which had grafts of various types, we have found the pattern of healing of great interest to us.

The authors stated that a new arterial wall was formed in the defect where the outer part of the media and adventitia was removed.

Does this imply that there was definite and irrefutable proof that new elastic tissue and smooth muscle were present? This, I think, is an important point.

In the quick-freezing of the portion of the wall, there was undoubtedly death of the tissue, considering the thickness that was frozen. Were any conclusions drawn from the various types of production of aneurysms as to which methods were most conducive to clot formation in the sac?

DR. BEN EISEMAN (Denver, Colo.).—My associate, Dr. Rainer, and I have been trying experimentally to produce aneurysms in dogs in a manner which we thought might duplicate that occurring following severe thoracic trauma. Our purpose was to determine whether aneurysms so produced in an undiseased aorta might not stabilize and not continue to expand. Some of our clinical evidence suggests that this might occur.

We opened the thoracic aorta immediately below the left subclavian takeoff and circumferentially incised the intima, media, and a fair portion of the adventitia. We followed these animals for months but only observed a slight swelling at the site of our circumferential incision without true aneurysmal dilatation.

I believe that the great value of experiments such as those reported by Dr. Economou is in clarifying the natural history of an aneurysm in an otherwise normal vessel—a project that we have attempted but without the success reported with the technique used by the authors.

DR. ECONOMOU (closing).—With respect to the best method of producing persistent experimental aortic aneurysms, we found that the most consistent was removal of the outer two-thirds of the media.

The photomicrographs of specimens prepared with Weigert's elastic tissue stain show that in aneurysms that repaired themselves elastic tissue is formed during the reparative process.

I believe that when Dr. Eiseman attempted to produce aneurysms by excising substantial amounts of the vessel wall from within, he really went deeper than in one facet of our experiments where only the intima and internal elastic membrane were excised. In effect, he substantiated our contention that, if subendothelial tissue is stimulated by injury, it triggers the repair of even substantial vessel wall defects.

In answer to Dr. Julian's question on the possibility of an intramural clot playing a role in the reparative process, I can only say that in aneurysms produced by transmural freezing, the intima remained intact, no mural clots were ever seen, and aneurysmal repair took place by subendothelial proliferation and differentiation.

FEMORAL BYPASS GRAFTS

EDWIN J. WHITMAN, M.D., JOSEPH M. JANES, M.D., JOHN C. IVINS, M.D., AND EINER W. JOHNSON, JR., M.D., ROCHESTER, MINN.

(From the Mayo Clinic and Mayo Foundation)

THE clinical use of grafts to bypass diseased segments of the femoral artery has become widely accepted in the past 5 years. Experimentally the procedure was attempted much earlier. Vesalius[36] in 1542 mentioned the insertion of a reed into the femoral artery of animals. The femoral artery was exposed, ligated, and incised, and a reed of the same caliber as the vessel was inserted into the proximal and distal ends of the artery. Ligatures then were cut, restoring pulsatile blood flow to the foot.

It is logical that the most favorable procedure to restore muscle function to normal is one that re-establishes arterial continuity. By means of a bypass vascular graft with an end-to-side anastomosis at each end, the arterial circulation[28] is re-established without danger of interrupting the collateral circulation.

The pathway to modern vascular surgery was established early in the 1900's by the work of Guthrie,[14, 15] Carrel,[4-6] and others.[26, 37] In fact vascular surgical techniques today differ little from the methods developed in the experimental laboratory 50 years ago. Over the years many materials have been used experimentally and clinically to replace blood vessels. These include arterial autografts, homografts[3] and heterografts,[7] autogenous and homologous veins,[4, 5, 11, 12, 25] glass,[30] vitallium,[2] stainless steel mesh,[27] and recently plastic materials, such as Ivalon, polyethylene, Orlon, nylon, Dacron, and Teflon. This imposing list testifies to the fact that a material suitable for replacing vessels has not been easy to find. Several of the plastic materials which have only recently come into use appear favorable, and only time will prove their ultimate worth.

Grafting of larger vessels, such as the abdominal aorta, has been satisfactorily accomplished regardless of the type of graft, although it now appears that a plastic prosthesis has many advantages over a homograft. The problem as far as smaller vessels are concerned has not been adequately solved. Several problems in grafting of small vessels will be mentioned.

Atherosclerosis, the underlying pathologic process, appears to be an important factor in the grafting of small vessels. Atherosclerosis is a process which involves many organs to various degrees as well as the vessel proximal and distal to a localized occlusion. The concept of "segmental occlusion" serves a valuable clinical purpose, but it is better to think of this as "multifocal" rather than segmental. The commonest reason for turning down a candidate for operation

Read at the Thirteenth Annual Meeting of the Society for Vascular Surgery, Atlantic City, N. J., June 7, 1959.

The Mayo Foundation, Rochester, Minnesota, is a part of the Graduate School of the University of Minnesota. Dr. Whitman is a Fellow in Surgery, Mayo Foundation, and Drs. Janes, Ivins, and Johnson are from the Section of Orthopedic Surgery, Mayo Clinic and Mayo Foundation.

is poor distal runoff due to extensive distal atherosclerotic involvement of the vessel. There must also be adequate flow through the proximal vessels, or good "run-in." During the surgical procedure thickened, friable vessels are encountered, and suturing through calcified plaques often is necessary. Areas of constriction, dilatation, and tortuosity of the distal part of the vessel cause eddy currents which may be a stimulus to early postoperative thrombosis. At present we have no means of modifying the pathologic process other than our palliative effort of bypassing diseased segments of vessels.

Another major problem is the size of the vessel and its relationship to the clotting mechanism. The body's amazing homeostatic mechanism protects the integrity of the healthy vascular tree down to the smallest capillary. This mechanism frequently fails in the presence of disease in the small vessels and certainly does not function adequately for vascular grafts in these small vessels. Experimental work has shown poor results in grafts less than 8 to 9 mm. in diameter. The diameters of most femoral arteries are in this range. The fibrin which forms an internal layer in the graft is fairly constant in thickness regardless of the diameter of the graft. The smaller the graft, the more important the role played by this fibrin lining. From studies of the fibrinolytic system it has been postulated that the endothelium plays a role in the activation of fibrinolysin. The endothelium as a living element has been found only in autogenous arterial grafts, and as a result of its absence fibrinolysis may be diminished in prosthetic grafts and therefore the chance of thrombosis increased. Unfortunately we have no control over the diameter of the vessel to be grafted.

The third factor, one over which we have some control, consists of the technical problems in this type of operation. These include surgical technique, the nature of the graft, and the use of ancillary pharmacologic agents. The surgical technique was accurately described by Carrel[4] 50 years ago, "fine silk, fine needle, Vaseline coating of the suture, accurate approximation of vessels, and perfect technique." Many pharmacologic agents are being developed, and some are now used in conjunction with vascular operations. Anticoagulants and fibrinolytic agents show promise. This leaves us with the problem of a suitable material for use as a graft and this will be discussed in this paper.

Bypass femoral grafts have been used at the Mayo Clinic since 1956. Homografts were used exclusively at first, but due to their limitations a better type of graft was sought. The crimped nylon prostheses as developed by Edwards and Tapp[13] appeared to offer many advantages, but late results proved unfavorable, and we have since switched exclusively to Teflon prostheses, with favorable results.

MATERIAL STUDIED AND METHODS OF EVALUATING RESULTS

This paper reviews the results of 96 femoral bypass grafts inserted in the 3-year period ending in October, 1958.

A total of 74 patients, 3 of whom were women, were treated. There were 58 homografts, 15 Edwards-Tapp nylon grafts, and 23 Teflon grafts.

Sixteen patients were 40 to 50 years of age and the majority of the patients were 51 to 60 years of age, with an average of 57 years (Fig. 1). This average does not include the 4 young patients who had grafts for trauma. The age period from 40 to 60 years is an active, productive period of life, and severe intermittent claudication can be totally disabling to an individual in this age group.

The grafts were evaluated clinically as functioning or nonfunctioning. The graft was considered nonfunctioning if there was minimal or no objective or subjective improvement. The functioning grafts were further considered as providing good or fair results. The result of the functioning graft was considered good if the patient was asymptomatic and if good peripheral pulses were present. It was considered fair when some improvement in symptoms and pulsation or both occurred, but the improvement was not of a maximal nature. A nonfunctioning graft in the immediate postoperative period is called an "acute failure," and one in the period of follow-up is called a "late failure" in this paper.

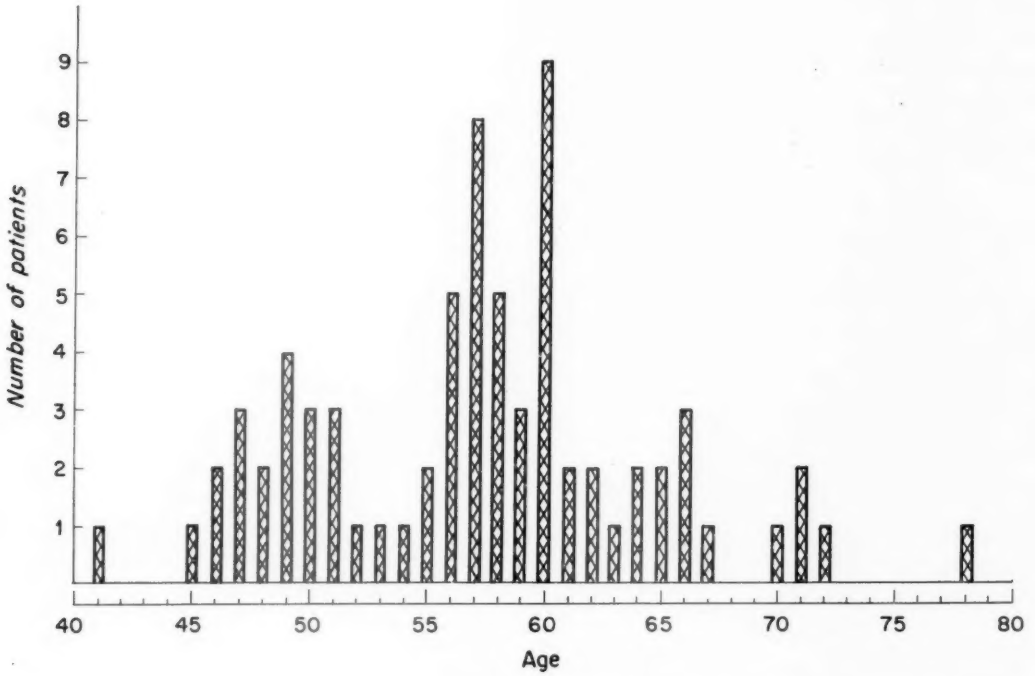


Fig. 1.—Age of patients having bypass graft in femoral artery; 4 cases of trauma in younger persons are excluded.

Almost every patient was evaluated initially by a member of the medical vascular service and by a vascular surgeon, and the majority were followed postoperatively and on subsequent visits to the clinic by the same team. Aortograms or femoral arteriograms or both were obtained on almost every patient before operation but were not made routinely after operation except in cases in which further operation was considered because of clinical failure of the original graft.

Follow-up letters were sent to all patients who had improved following operation. We asked about walking distance, warmth of feet, soreness, pain or numbness of feet, and further surgical procedures. Besides follow-up letters most of these patients have been examined at the clinic one or more times since operation.

The patient's ability to walk without claudication, objective evidence of good peripheral pulses, and the finding of a patent graft on arteriography show excellent correlation. Intermittent claudication is a pathognomonic symptom in occlusive disease and usually

does not occur until the area of the lumen of the femoral artery is reduced to 20 per cent or less of normal. The area of the lumen can be reduced 50 per cent without any change in flow.[19, 29] Intermittent claudication returned in all except one patient in whom the graft failed. The patient who was the exception had had a graft inserted because of trauma when he was 12 years of age. The graft failed and no peripheral pulses are present, but the patient can walk 10 miles without claudication. Adequate collateral circulation apparently developed; it is not uncommon for young persons to be relatively asymptomatic following occlusion of the femoral artery. Conversely, one patient had pulses restored after operation but did not have any relief from claudication. This evidence seems to us to provide ample justification for evaluating the results of our grafts clinically.

INDICATIONS FOR USE OF THE BYPASS GRAFT

The indications for operation are given in Table I. All patients with segmental occlusion of the superficial femoral artery who had operations had relatively severe claudication before operation. Some patients also had ischemic neuropathy, ulcers of toes, and at times pregangrenous and gangrenous changes. Most of the patients were in good general health, although some of the ones operated upon in the early period had complicated vascular problems.

TABLE I. INDICATIONS FOR FEMORAL BYPASS GRAFT

INDICATIONS		GRAFTS
Arteriosclerosis obliterans with segmental occlusion of the superficial femoral artery		76
Unilateral	52	
Bilateral (12 cases)	24	
Popliteal aneurysm		7
Femoral aneurysm		1
Regrafting following failure of homograft		8
Trauma to femoral artery		4
Total		96

The findings of aortography or arteriography were considered important in deciding operability. The major factor in operability was a segmental lesion with good distal runoff (Fig. 2). We also looked for patency of the deep femoral artery. Operation was carried out on only one patient in our series in the presence of occlusion of the deep femoral artery, and the result was poor.

In selecting patients for operation, we have had to keep in mind that a graft may be of only palliative value, but an extra few years of moderate activity, especially for a patient in the active years of life, may be invaluable.

FEMORAL HOMOGRAFTS

The arterial homograft was the first type of vessel graft to be used extensively in our clinical practice.[9] Homografts appear to function as conduits which carry blood, but they are never truly integrated by the host tissue.[1, 3, 31] Guthrie concluded in 1919, "as to the use of homografts, 'To restore and maintain mechanical function, such an implanted segment need only temporarily restore mechanical continuity and serve as a scaffolding or bridge for the laying down of an ingrowth of tissue derived from the host.'" This fact has been well substantiated by the recent work of Szilagyi and co-workers.[34] Homografts

will show signs of degenerative processes including aneurysmal dilatation (Fig. 3), tortuosity, and roughening of the fibrin network or so-called pseudo-intima. It appears in some cases that a homograft fails because of an immunologic response by the host. Some investigators[32] have stated that viability of a homograft is important for prolonged function, but there is no conclusive evidence for this.

A. *B.*

Fig. 2.—*A* and *B*, Segmental occlusion of superficial femoral artery. Femoral arteriogram shows patency of the deep femoral artery and good runoff distally. Ideal candidate for femoral bypass graft.

Homografts have several disadvantages: (1) they degenerate as a function of time, (2) an adequate supply is not readily available, (3) they present problems in storage, preservation, and sterilization, and (4) one cannot always choose a proper size in each case. In view of these disadvantages a better type of material for grafting was sought. This led to the development of the plastic prosthesis.

Homografts, however, offer certain advantages over plastic grafts, namely, ease of suturing and minimal loss of blood at operation both through the graft and at the anastomotic line. Some workers add, too, that there is experimental proof that a homograft is more satisfactory than the plastic graft in small vessel grafting.

Fifty-eight homografts were inserted into 46 patients without any deaths. Seven of these patients had bilateral homografts. Eight required subsequent grafts after failure of the original homograft. Four of these 8 patients had a second homograft inserted and 4 had plastic prostheses. One had another graft inserted distally to the original graft. Operations on 38 patients were done because of segmental occlusion of the superficial femoral artery. Five grafts were for popliteal aneurysms and three for trauma. Aortic or aortoiliac grafts had been inserted in 6 of the 46 patients prior to, or at the time of, femoral grafting. The majority of the grafts were inserted in an end-to-side fashion from the common femoral to the popliteal artery. For several of the popliteal aneurysms, however, grafts were inserted in end-to-end fashion from the superficial femoral to the distal part of the popliteal artery.

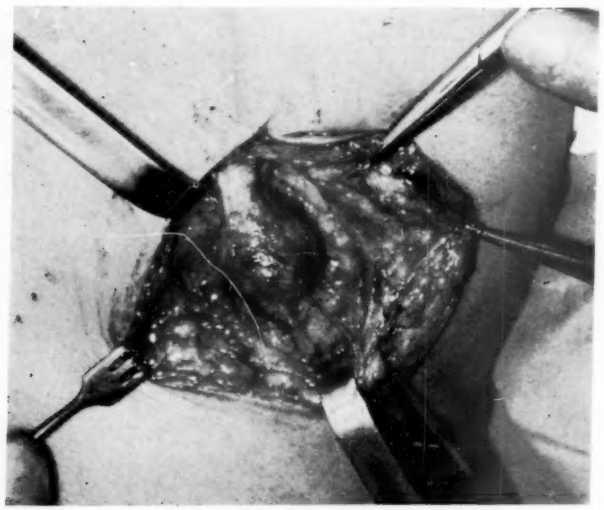


Fig. 3.—True aneurysm in homograft at junction of anastomoses between 2 separate homografts required, 4 months after insertion.

Fourteen of the 58 grafts were acute failures, that is, they failed to function in the operative or immediate postoperative period. The commonest cause of acute failure was extensive atherosclerosis, which accounted for 9 failures. Two failures were directly attributed to homografts, one was due to postoperative heparinization, and 2 were of undetermined nature (Table II).

In the group of patients having homografts 6 had associated aortic grafts: 2 for aortic aneurysm and 4 for aortoiliac occlusive disease. Five of these patients had acute failures of the femoral graft. The other patient died 7 months after operation of hypertension secondary to thrombosis of the renal artery presumably following aortic operation. The femoral graft was patent at postmortem examination. Infection and hemorrhage accounted for three

TABLE II. ACUTE FAILURE OF FEMORAL HOMOGRAFTS

CASE	AGE AND SEX	DETAILS
		Atherosclerosis and aortic grafts
1	56 M	Aneurysm of abdominal aorta, right iliac and right femoral artery; aortic homograft 10 weeks prior to femoral graft; fat necrosis of femoral incision with failure of graft
2	50 M	Aortic homograft inserted for iliac occlusion at the same time as femoral graft; the latter functioned until aortic graft ruptured 1 week after operation; another homograft was inserted in aorta; a second femoral homograft inserted 3 months later gave a fair result until patient died 3 months later
3	57 M	Aortic homograft inserted for aneurysms of aorta and iliac artery; bypass for popliteal aneurysm performed 7 months later; postoperative infection and hemorrhage required ligation of femoral graft
4	51 F	Ischemia in foot followed insertion of aortic graft for aortoiliac occlusion; femoral homograft as an emergency procedure; graft failed and an amputation was required 2 weeks later
5	71 M	Aortic homograft for aneurysm of abdominal aorta and iliac artery; a femoral graft was inserted 2 weeks later; postoperative abscess required ligation of graft; eventual amputation
		Atherosclerosis and ischemic neuropathy
6	60 M	Ischemic neuropathy and ulceration before insertion of graft; severe postoperative pain required amputation 10 weeks later
7	62 M	Ischemic neuropathy; no improvement after insertion of the femoral graft; amputation 24 months later
		Atherosclerosis and poor distal runoff
8	41 M	
9	51 M	Satisfactory graft of contralateral extremity
		Failure of homograft
10	46 M	Graft inserted for traumatic aneurysm of femoral artery; fusiform dilatation and rupture of graft 19 days after operation; amputation above the knee 20 days later
11	58 M	Homograft occluded at the conclusion of surgical procedure
		Heparinization
12	57 M	Heparin given continuously by vein after operation; hematoma compressed graft and caused occlusion; thromboendarterectomy with poor results; a subsequent femoral graft inserted elsewhere gave a good result
		Cause not determined
13	47 M	Improved pulsation but no change in claudication after operation
14	58 F	No improvement after operation

of the failures of the femoral grafts in the group who also had aortic grafts. Infection did not occur in any other patient having homografts inserted, and we must assume that in spite of functioning aortic grafts the blood supply to the region of the femoral graft was compromised. The other 2 failures were the immediate result of complications of aortic surgery.

We recently have reconsidered our criteria for operability of patients with aortoiliac occlusion and femoral occlusion. At present we do not insert a femoral graft in the presence of uncorrected aortoiliac occlusion. An

abdominal aneurysm certainly warrants grafting even in the presence of distal femoral occlusion, but occlusion of the femoral artery is not an absolute indication for grafting.

Some surgeons have considered ischemia and neuropathy as contraindications of femoral grafts. Although two of the acute failures were associated with these conditions, other patients with them have been relieved of symptoms after grafting.

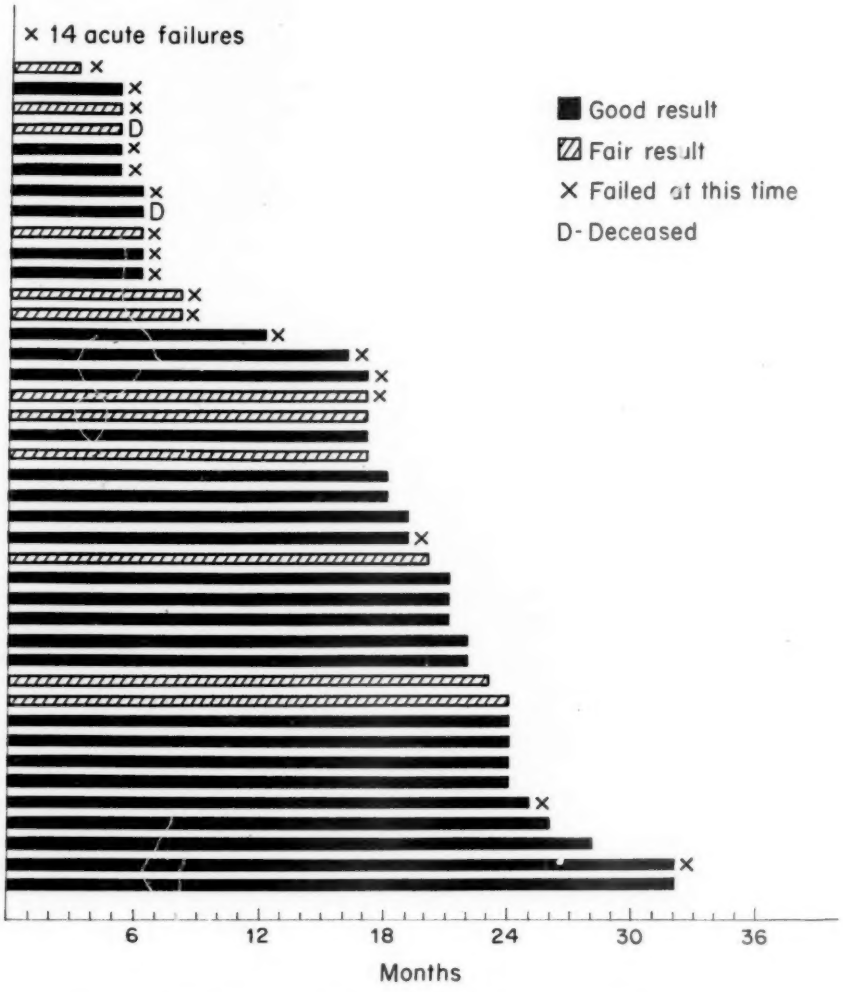


Fig. 4.—Longevity of femoral homografts in 55 cases.

Inherent defects in homografts accounted for 2 acute failures and 5 late failures.

Six of the patients who had acute failures are known to have had eventual amputation, most of them above the knee.

Late Results.—Of the 58 homografts inserted, 44 functioned beyond the early postoperative period (Table III and Fig. 4). The function of 33 was considered good and that of 11 fair at time of dismissal. Two patients who died

TABLE III. STATUS OF 58 HOMOGRAFTS

IMMEDIATE POSTOPERATIVE PERIOD	NUMBER	PER CENT
Acute failure	14	24
Functioning	44*	76

DURATION OF FUNCTION OF 53 GRAFTS*

TO TIME OF FOLLOW-UP	NUMBER	TO TIME OF FAILURE	NUMBER
16-20 mo.	7	Acute failure	14
21-24 mo.	11	< 6 mo.	9
25-32 mo.	3	7-12 mo.	3
		13-18 mo.	3
		>18 mo.	3
Total	21	Total	32
Per cent	40	Per cent	60

*Three patients lost to follow-up. Two patients died shortly after operation; although grafts were functioning at time of death, they were excluded because of short follow-up.

in the year after operation have been excluded from the follow-up series although they had functioning grafts at the time of death. Follow-up data were available concerning 39 of the remaining 42 grafts. The follow-up period was 16 to 32 months.

Eighteen of the 39 grafts failed in the follow-up period. Six of these were considered to give fair results after operation. Two patients had grafts which failed bilaterally. Thrombosis accounted for 12 failures from 3 to 32 months after operation. Five failures were due to failures of the graft, and one was due to infection following insertion of a popliteal graft 8 months after insertion of the femoral graft.

Second grafts were inserted into 4 patients who had thrombosis: 2 homografts, 1 nylon graft, and 1 Teflon graft. At time of follow-up 2 of these were functioning: a homograft for 23 months and a Teflon for 7 months. Of the 5 late failures due to homograft failure, 3 were due to true aneurysms and 2 to false aneurysms. Three of these patients had second grafts inserted; the one Teflon graft inserted is functioning 12 months after operation.

Seven failures (22 per cent of all failures to date in homografts) were due exclusively to failure of the homograft. We feel that these complications could have been avoided with plastic prostheses. One-half of the homografts that have failed in the late period failed in the first 6 months after operation. Of 30 grafts which functioned at least 6 months, 9 failed from 7 to 32 months after operation (Fig. 4 and Table III).

At the time of follow-up 21 homografts were known to be functioning. Sixteen were considered as having good function and 5 as fair. At a period 18 to 32 months following operation more than one-third of the homografts were functioning. No grafts which were considered poor or fair initially showed later improvement. More than one-half the grafts originally considered fair have since failed.

Comment.—It appears important to evaluate the patient in regard to other peripheral vascular lesions. Aortograms and arteriograms are important in adequately evaluating the vascular tree. Surgery should be offered to patients with a good history of intermittent claudication, good runoff as seen on arteriograms, and absence of more severe lesions such as aortic aneurysms which require immediate operation.

Many factors must be considered in evaluating these grafts. The extent of atherosclerosis appears to be the most important factor in successful grafting. Patients having aortic surgery as well as femoral surgery had poor results from femoral grafts. Patients with preoperative ischemia presenting with neuropathy and pregangrenous ulcers also had poor results. The patients we saw with trauma were seen late in their course and consequently had poor results.

We must also remember that many of the grafts which ultimately failed gave relief of symptoms for 3 to 32 months. In view of the inherent disadvantages of homografts, a better type of graft appeared desirable[10, 13, 21, 35] and this led at the end of 2 years to use of plastic prostheses for replacement of vessels.

NYLON ARTERIAL GRAFTS

The factors taken into consideration in selecting an ideal plastic prosthesis[21, 23] were availability, flexibility, inertness, durability, ease of handling, cost, ease of sterilization, and hemodynamic properties. No graft appeared to exhibit all of these qualities, but the crimped nylon graft as developed by Edwards and Tapp appeared to possess many of them.

Fifteen Edwards-Tapp nylon grafts were inserted as femoral bypass grafts in an end-to-side fashion. Indications for 11 of the nylon grafts were segmental occlusion of the superficial femoral artery; for 3, failure of a homograft; and for 1, traumatic laceration of the femoral artery. One of the 15 patients (Table IV) had a homograft inserted in the contralateral extremity. There were 4 acute failures of nylon grafts.

TABLE IV. ACUTE FAILURES OF NYLON AND TEFLON GRAFTS

CASE	AGE AND SEX		PERTINENT DETAILS
			Nylon grafts
19	3	F	Fracture of femur and laceration of femoral artery; progressive ischemia following grafting requiring amputation above the knee 1 week later
20	27	M	Thrombosis of homograft 8 months after insertion for trauma; nylon graft occluded in 2 weeks with increasing ischemia; amputation above knee was necessary 1 week later; pathologic report indicated rejection of nylon graft; a fluid-filled channel surrounded the graft and numerous foreign body giant cells were present in surrounding tissue
21	55	M	False aneurysm developed in homograft 16 months following insertion; nylon graft inserted and wound infection developed after operation; infection could not be controlled and graft was finally removed after 6 months; amputation above the knee was necessary 1 week later
22	52	M	Occlusion of right common iliac artery and right superficial femoral artery; iliopopliteal graft; no postoperative improvement
			Teflon grafts
23	60	F	Diabetic, pregangrenous lesion of foot, arteriogram indicated disease at the level of the popliteal trifurcation but patient desired surgery in an attempt to prevent major amputation; there was a weak pulse following surgery with gradual healing of foot for several weeks; following this there were progressive ischemia and wound infection, and amputation below the knee was necessary 6 weeks after grafting
24	61	M	No postoperative improvement subjectively or objectively

One patient (Case 19) had a severe soft tissue injury as well as a comminuted fracture of the femur and laceration of nerves and vessels. Grafting was attempted in the hope of saving the limb. Two patients had failures of previous grafts, and nylon grafts were inserted to re-establish circulation. Although all 3 cases ended in amputation, this likely would have been the ultimate outcome if no further operation had been performed. In one case (Case 20) there was outright rejection of the nylon graft by the host. We have not seen this in any other failures.

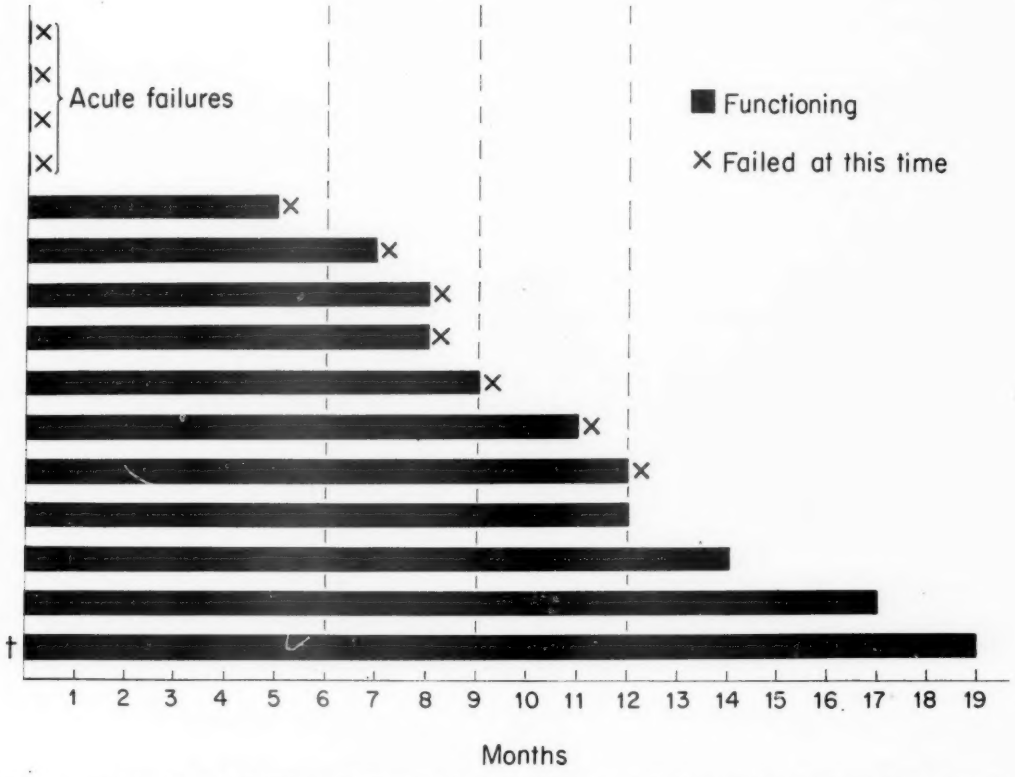


Fig. 5.—Longevity of 15 Edwards-Tapp nylon prostheses. † indicates that patient has a homograft in contralateral extremity.

The remaining 11 grafts were considered as giving good functional results in the immediate postoperative period (Table V). To our dismay 7 of these grafts had failed by the time of follow-up 5 to 16 months later (Table V and Fig. 5). Four grafts were functioning at 12, 14, 17, and 19 months, respectively, after operation. Selection of patients for operation was based on the same standards as those for patients who later had Teflon grafts, and many were better candidates than the patients having homografts. Yet our failures were much higher in this group than in the groups with homografts and Teflon grafts.

All of the failures in the late period were due to thrombosis.[33] Many of these failures occurred in an acute episode which was not the rule in homograft failures. Two patients noted acute return of symptoms following squatting. One of these patients was taking anticoagulants at time of failure. The

TABLE V. STATUS OF 15 NYLON GRAFTS

IMMEDIATE POSTOPERATIVE PERIOD	NUMBER	PER CENT
Acute failure	4	27
Functioning	11	73

DURATION OF FUNCTION			
TO TIME OF FOLLOW-UP	NUMBER	TO TIME OF FAILURE	NUMBER
12 mo.	1	Acute failure	4
14 mo.	1	<6 mo.	1
17 mo.	1	7- 9 mo.	4
19 mo.	1	10-12 mo.	2
Total	4	Total	11
Per cent	27	Per cent	73

other patient was having a checkup at the clinic. He was asymptomatic and had good peripheral pulses. He squatted in his hotel room and noted sudden coldness and pain in the leg. An arteriogram the next day indicated complete occlusion of the femoral and popliteal arteries. Another patient struck his knee; acute symptoms followed this slight trauma.

Comment.—After we first began to use nylon grafts, abundant experimental evidence, mainly by Harrison and co-workers,[16-18] showed that nylon has properties which make it undesirable for use as a long narrow prosthesis.[8] Nylon is a condensation product from which water has been expelled by polymerization. Hydrolysis may be expected to occur in it when it is exposed to body fluids for a long period. This has been seen in failure and dissolution of nylon used in prosthetic heads of femurs. Nylon is one of the more wettable materials used for grafts, absorbing 4.5 per cent water as compared to Teflon which absorbs less than 0.4 per cent. Anticoagulant power of a surface in vitro has been seen to be inversely proportional to force of adhesion between surface and water.[20] There is also evidence that reduction in tensile strength and stability may be related to absorption of water. Studies have shown that 83 per cent of the tensile strength of the nylon graft is lost in 6 months after implantation.

Reaction of the tissue to nylon is great, both early and late after implantation. Healing is delayed; a thick fibrous enclosure forms on the outside of the graft, and a fibrin layer 2 to 3 mm. thick forms on the inside. It takes months for the internal layer of fibrin to become firmly adherent to the graft. This may well account for the acute failures following squatting. Perhaps the fibrin lining breaks loose and occludes the graft. These factors may well eliminate the advantage of nonbuckling of crimped grafts. Certainly the thick fibrin layer in nylon grafts is not conducive to prolonged function of a graft of small diameter and may enhance the chance of early thrombosis.

Perhaps, too, the thick fibrous external layer around a nylon graft associated with decrease of tensile strength may allow the surrounding tissues to exert enough pressure to cause partial collapse of the graft and this may increase the likelihood of thrombosis.

These facts based on experimental studies and our clinical experience indicate that nylon is an unsatisfactory material for femoral bypass grafts.

TEFLON GRAFTS

Teflon, as opposed to nylon, has been proved experimentally and clinically to be an ideal material for femoral grafts.[16-18, 35] Teflon causes only minimal tissue reaction and healing occurs rapidly after implantation. Teflon is inert, is unaffected by body fluids, and is chemically resistant to all known substances. Water absorption is less than 0.4 per cent, and only a thin fibrous enclosure forms externally and a thin fibrin layer internally. There is no loss of tensile strength after implantation. Teflon is easy to handle and cut and comes in woven or knitted form. The woven graft does not require preclotting or heat sealing of ends to prevent fraying.

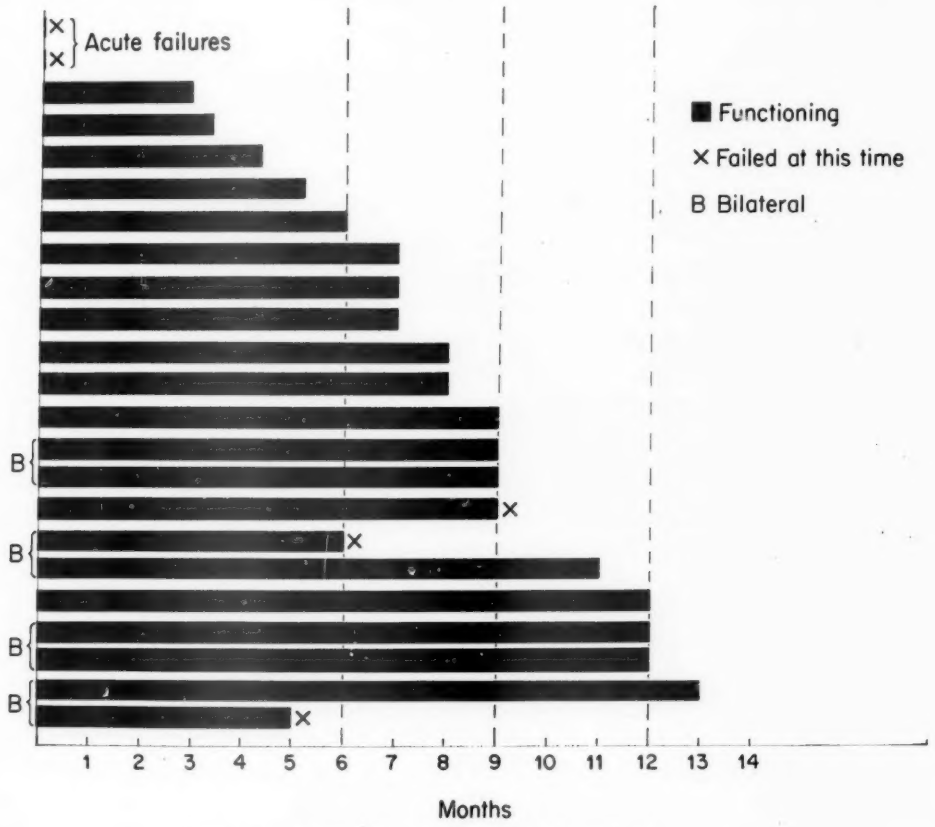


Fig. 6.—Longevity of 23 Teflon prostheses.

Twenty-three Teflon grafts have been inserted into 19 patients. Nineteen grafts were for segmental occlusion of the superficial femoral artery, for which 4 patients had bilateral Teflon grafts. Two grafts were for popliteal aneurysm and 2 for failure of homografts. We were able to obtain follow-up information 3 to 10 months after operation on all patients (Table VI and Fig. 6). There were 2 acute failures (Table IV).

Of the 21 functioning grafts after operation all were considered as giving good results at time of dismissal. There have only been 3 late failures of Teflon grafts. Two late failures were in patients who had functioning grafts in the contralateral extremity. Both had incisional infections in the groin

TABLE VI. STATUS OF 23 TEFLON GRAFTS

IMMEDIATE POSTOPERATIVE PERIOD	NUMBER	PER CENT
Acute failure	2	9
Functioning	21	91

DURATION OF FUNCTION			
TO TIME OF FOLLOW-UP	NUMBER	TO TIME OF FAILURE	NUMBER
3-6 mo.	5	Acute failure	2
7-9 mo.	8	5 mo.	1
>9 mo.	5	6 mo.	1
		9 mo.	1
Total	18	Total	5
Per cent	78	Per cent	22

after insertion of the Teflon graft. A low grade infection persisted in one patient for 5 months at which time the graft became occluded. The patient was asymptomatic as far as claudication was concerned until the graft failed. In the other patient dehiscence of the groin incision occurred, but it eventually healed and the graft functioned for 6 months. It then became occluded. Increased fibrosis secondary to the infection may have played a part here. The condition of each of these patients is no worse than prior to operation and neither is in immediate danger of amputation. This is in striking contrast to patients with homografts and nylon grafts who required amputation shortly after failure of grafts due to infection. It also indicates that Teflon grafts will function for long periods in spite of the presence of infection.

Two of the functioning grafts are in patients who previously had had homografts which failed. One of these patients had had 2 previous homografts with the development of true aneurysms in each. The Teflon graft in this patient is functioning 12 months after operation. In the other patient also the Teflon graft was inserted after an aneurysm in a homograft; the Teflon graft is functioning well at 7 months.

Comment.—In spite of the small series of nylon and Teflon grafts the present comparison appears significant. There have been only 3 late failures in 21 Teflon grafts and 7 late failures in 11 nylon grafts. Thirteen out of 14 of the Teflon grafts for which the follow-up period is 6 months or longer are functioning. All late failures of nylon grafts occurred between 5 and 12 months after operation (Fig. 6). Comparison of the late failures of Teflon grafts with those of homografts (three-fourths of which occurred in the first 6 months after insertion) and those of nylon (two-thirds of which occurred by 9 months) encourages us. There is reason to believe that the longer a graft functions the more likely it is to remain functioning.[22] This appears true for homografts, and possibly this is survival of the fittest as far as homografts are concerned. Time alone will prove whether this is true for Teflon grafts also. To date we are well satisfied with the Teflon graft.

POPLITEAL ANEURYSM

Seven bypass grafts for popliteal aneurysm are included in this series. Five patients had homografts and 2, Teflon grafts. Our treatment as reported in 1951 by two of us (J. M. J. and J. C. I.)[24] previously consisted of ligation of

the aneurysm accompanied by lumbar sympathectomy. This gave satisfactory results, but with the advent of suitable grafts for vessels of this size, it was decided that re-establishment of arterial continuity would be more satisfactory when possible.

Depending on the circumstances, we resect or ligate the aneurysm and insert a graft. We still perform lumbar sympathectomy in the majority of cases. As yet we do not have enough cases to evaluate the worth of sympathectomy. The one acute failure in this group occurred in a patient who had had an aortoiliac homograft for aneurysms of the aorta and iliac vessel on the same side 7 months previously. An infection followed the operation for popliteal aneurysm, and the graft had to be ligated. One patient has been lost to follow-up. The other 5 patients have had good results to date, 6 to 28 months after operation (Table VII).

TABLE VII. POPLITEAL ANEURYSMS

AGE AND SEX	PROCEDURE	TYPE OF GRAFT	FOLLOW-UP (MO.)	STATUS
57 M	Excision with insertion of bypass graft 7 months after aortic and iliofemoral homografts for multiple aneurysms; postoperative infection and hemorrhage requiring ligation of graft	Homograft	Acute failure	Poor
78 M	Bypass graft with lumbar sympathectomy	Homograft	None	?
70 M	Bypass graft for femoral and popliteal aneurysms	Homograft	24	Good
67 M	Bypass graft	Homograft	28	Good
59 M	Bypass graft with lumbar sympathectomy	Homograft	23	Good
63 M	End-to-end graft after resection of popliteal aneurysm	Teflon	7	Good
56 M	Bypass graft with lumbar alcohol block	Teflon	6	Good

COMMENT AND SUMMARY

Femoral bypass grafts have proved valuable in the treatment of peripheral arteriosclerosis. Follow-up information was available concerning 91 of the 96 grafts inserted at the Mayo Clinic from 1956 to October, 1958. Of these, 43 were functioning grafts at time of follow-up. Eighteen of the 23 Teflon grafts (78.3 per cent) were functioning. Teflon is far superior to any other type of graft in our experience. Many patients who had grafts which eventually failed had palliation for a considerable period before ultimate failure, some up to 32 months. We have had no surgical mortality and little morbidity. Ten patients eventually have had amputations. More than one-half of the 10 patients would have required immediate amputation if grafting had not been performed. It is hard to judge from this study how many patients would have required amputation without grafting. Other patients who now have functioning grafts have undoubtedly been saved from amputation or have required only amputations of single gangrenous toes following grafting. Many of our patients were 40 to 60 years old, with the average age being 57 years. These patients are in a productive age group, and many are severely handicapped by intermittent claudication and manifestations of ischemia. The

results of the study suggest that, with proper indications, the use of femoral bypass grafts for occlusive disease of the femoral artery is worth while. In our hands Teflon is the material of choice.

ADDENDUM

Since this paper was submitted for publication, a total of 52 Teflon prostheses have been used as femoral bypass grafts. These were used in a total of 42 patients. Eight patients had bilateral grafts and 3 required second grafts for failure of the original. For the past 10 months woven Teflon grafts have been used exclusively. Loss of blood was reduced to a minimum by the use of the woven prosthesis which does not require preclotting and which does not leak except for mild "sweating" when clamps are released.

In this group there were 5 acute failures, an acute failure rate of 10 per cent, and 10 additional failures in the late period. At this time more than 70 per cent of all Teflon prostheses are functioning, and the longest follow-up is now 19 months.

REFERENCES

1. Bencini, A., and Bellinazzo, P.: Experimental Studies of the Fate of Arterial Homografts, Angiology **4:** 483-495, 1953.
2. Blakemore, A. H., and Lord, J. W., Jr.: A Nonsuture Method of Blood Vessel Anastomosis: Experimental and Clinical Study, J. A. M. A. **127:** 685-690, 1945.
3. Brown, R. B., Huggins, C. E., and Koth, D. R.: An Experimental Revaluation of the Problem of Small Vessel Replacement, SURGERY **43:** 63-75, 1958.
4. Carrel, A.: Surgery of the Blood-Vessels and Its Application to the Changes of Circulation and Transplantation of Organs, Bull. Johns Hopkins Hosp. **17:** 236-237, 1906.
5. Carrel, Alexis: Anastomosis and Transplantation of Blood-Vessels, Am. Med. **10:** 284-285, 1905.
6. Carrel, Alexis: Ultimate Results of Aortic Transplantations, J. Exper. Med. **15:** 389-392, 1912.
7. Creech, Oscar, Jr., De Bakey, M. E., Self, Milton, and Halpert, Béla: The Fate of Heterologous Arterial Grafts: An Experimental Study, SURGERY **36:** 431-444, 1954.
8. Creech, Oscar, Jr., Deterling, R. A., Jr., Edwards, Sterling, Julian, O. C., Linton, R. R., and Shumacker, Harris, Jr.: Vascular Prostheses: Report of the Committee for the Study of Vascular Prostheses of the Society for Vascular Surgery, SURGERY **41:** 62-80, 1957.
9. De Bakey, M. E., Crawford, E. S., Creech, Oscar, Jr., and Cooley, D. A.: Arterial Homografts for Peripheral Arteriosclerotic Occlusive Disease, Circulation **15:** 21-30, 1957.
10. Deterling, R. A., Jr., and Bhonslay, S. B.: An Apprasial of Woven Synthetic Prostheses in the Vascular System, A. M. A. Arch. Surg. **72:** 76-91, 1956.
11. Dye, W. S., Grove, W. J., Olwin, J. H., and Julian, O. C.: Two-to-Four-Year Behavior of Vein Grafts in the Lower Extremities, A. M. A. Arch. Surg. **72:** 64-68, 1956.
12. Dye, W. S., Olwin, J. H., and Julian, O. C.: Further Considerations on the Indications for and Limitations of Direct Surgery in Arteriosclerosis, Circulation **8:** 708-714, 1953.
13. Edwards, W. S.: Plastic Arterial Grafts, Springfield, Ill., 1957, Charles C Thomas, pp. 126.
14. Guthrie, C. C.: Blood-Vessel Surgery and Its Applications, New York, 1912, Longmans, Green & Co., pp. 360.
15. Guthrie, C. C.: Survival of Engrafted Tissues. III. Blood-Vessels, Heart **2:** 115-122, 1910.
16. Harrison, J. H.: Synthetic Materials as Vascular Prostheses. I. A Comparative Study in Small Vessels of Nylon, Dacron, Orlon, Ivalon Sponge and Teflon, Am. J. Surg. **95:** 3-15, 1958.
17. Harrison, J. H.: The Use of Teflon as a Blood Vessel Replacement in Experimental Animals, Surg. Gynec. & Obst. **104:** 81-87, 1957.
18. Harrison, J. H., Swanson, D. S., and Lincoln, A. F.: A Comparison of the Tissue Reactions to Plastic Materials, A. M. A. Arch. Surg. **74:** 139-144, 1957.
19. Herrick, J. F., and Baldes, E. J.: The Thermo-stromuhr Method of Measuring Blood Flow, Physics **1:** 407-417, 1931.
20. Hirschboeck, J. S.: Delayed Blood Coagulation in Methyl Methacrylate (Boilable "Lucite") Vessels, Proc. Soc. Exper. Biol. & Med. **47:** 311-312, 1941.

21. Hohf, R. P., Trippel, O. H., and Laufman, Harold: Clinical Comparison of Homografts and Braided Nylon in Arterial Bypass Surgery, A. M. A. Arch. Surg. **77**: 552-560, 1958.
22. Hoye, S. J., and Warren, Richard: Follow-up Studies of Iliofemoral Arterial Reconstruction in Arteriosclerosis Obliterans, New England J. Med. **254**: 102-106, 1956.
23. Hufnagel, C. A.: The Use of Rigid and Flexible Plastic Prostheses for Arterial Replacement, SURGERY **37**: 165-174, 1955.
24. Janes, J. M., and Ivins, J. C.: A Method of Dealing With Arteriosclerotic Popliteal Aneurysms, SURGERY **29**: 398-406, 1951.
25. Julian, O. C., Dye, W. S., Jr., Olwin, J. H., and Jordan, P. H.: Direct Surgery of Arteriosclerosis, Ann. Surg. **136**: 459-474, 1952.
26. Klotz, Oskar, Permar, H. H., and Guthrie, C. C.: End Results of Arterial Transplants, Ann. Surg. **78**: 305-320, 1923.
27. Lary, B. G., Meine, E. L., Jr., and de Takats, Geza: The Experimental Use of Steel Mesh Tubes for the Replacement of Arterial Segments, A. M. A. Arch. Surg. **72**: 69-75, 1956.
28. Linton, R. R., and Menendez, C. V.: Arterial Homografts: A Comparison of the Results With End-to-End and End-to-Side Vascular Anastomoses, Ann. Surg. **142**: 568-585, 1955.
29. Mann, F. C., Herrick, J. F., Essex, H. E., and Baldes, E. J.: The Effect on the Blood Flow of Decreasing the Lumen of a Blood Vessel, SURGERY **4**: 249-252, 1938.
30. Murray, Gordon, and Janes, J. M.: Prevention of Acute Failure of Circulation Following Injuries to Large Arteries: Experiments With Glass Cannulae Kept Patent by Administration of Heparin, Brit. M. J. **2**: 6-7, 1940.
31. Parsons, H. G., Gerbode, Frank, and Cox, A. J.: Studies in Aortic Autografts and Homografts: Do Homografts Survive? Angiology **3**: 306-316, 1952.
32. Peirce, E. C., II: The Use of Viably Preserved Tissue for Homologous Arterial Graft, Ann. Surg. **136**: 228-238, 1952.
33. Shaw, R. S., and Wheelock, Frank: Blood Vessel Grafts in the Treatment of Chronic Occlusive Disease in the Femoral Artery, SURGERY **37**: 94-104, 1955.
34. Szilagyi, D. E., McDonald, R. T., Smith, R. F., and Whitcomb, J. G.: Biologic Fate of Human Arterial Homografts, A. M. A. Arch. Surg. **75**: 506-529, 1957.
35. Szilagyi, D. E., Shonnard, C. P., Lopez, Y., Lopez, Juan, and Smyth, N. P. D.: The Replacement of Long and Narrow Arterial Segments: An Experimental Study of Heterografts and Seamless Woven Nylon and Teflon Prostheses, SURGERY **40**: 1043-1059, 1956.
36. Vesalius, Anders: On Dissection of the Living: What Might Be Learned by Dissection of the Dead and What of the Living, 1542.
37. Watts, S. H.: The Suture of Blood Vessels: Implantation and Transplantation of Vessels and Organs: An Historical and Experimental Study, Bull. Johns Hopkins Hosp. **18**: 153-179, 1907.

DISCUSSION

DR. JERE W. LORD, JR. (New York, N. Y.).—The careful and detailed analysis of the results of homograft and two types of plastic prostheses is most worth while.

I would like to put in a plug for the autologous vein graft. Not only is the greater saphenous vein available in most patients as a graft whose good features include sterility, lack of antigenic properties, and the ability to bend and angulate in the region of joints, but also usually is of appropriate size for peripheral arterial replacement or bypass. Occasionally it is too small, previously thrombosed, or excised, and a more common criticism is that it may be too short.

In a group of 30 patients with acute traumatic arterial defects, neoplasms requiring resection of a segment of artery, with chronic arteriovenous fistula and aneurysm, there has not been a single failure either due to thrombosis or aneurysm formation. On the other hand, in a group of approximately 15 patients with vein graft bypass for segmental occlusive disease, 6 have failed. This experience compares favorably with the result of Dr. Janes's Teflon grafts. Our own experience with Teflon grafts has not been as happy.

CUFFLESS, NONCANNULA, CONTINUOUS RECORDING OF BLOOD PRESSURE

Statement of Concept and Description of Methods for Measurement Based on Principle of Capacitance

Ralph Adams, M.D.,* and Robert W. Corell, B.S.,** Wolfeboro, N. H.

(From the Department of Surgery, Boston University School of Medicine; the Department of Mechanical Engineering, Massachusetts Institute of Technology; and the Division of Surgery, Huggins Hospital)

THE accurate recording of human blood pressure has achieved new importance with the developments of modern surgery, the advent of space medicine, and the recognition of close interrelationships between blood pressure, medication, and psychosomatic or physical status. These developments have prompted re-evaluation of the basis for obtaining creditable information regarding arterial tension and have led to recognition of the following four facts regarding blood pressure determination. First, all of the presently available indirect methods of recording blood pressure are based on arterial constriction, and at end-point are identical to the clinically applied method of sphygmomanometry. Second, most cannulation methods are capable of yielding accurate clinical and research data, but are cumbersome and slightly hazardous. Third, these two techniques have been studied until it would appear that little is to be gained in further attempts to improve them. Fourth and finally, if there are advancements to be made in ease or accuracy of blood pressure recording, it seems as though some new approach to the problem must be found.

Numerous potential solutions to the problem of indirect blood pressure measurement were examined. Photoelectric techniques, as well as plethysmographic procedures, have proved to be invalid as methods for blood pressure determination. Pressure perturbation, or ultrasonic techniques, may have some potential in this area. King[6] found that the wave propagation speed of the medium within the cardiovascular system is theoretically a function of mean blood pressure. Experimental evidence[3] has indicated that the propagation velocity varies in a quasilinear manner with mean pressure. The resolution of this variance would appear to be quite good, that is, 25 per cent propagation speed variation for 50 mm. Hg mean blood pressure change. Unfortunately, the concept is attended by serious practical limitations, such as need for arterial exposure.

Supported in part by grant-in-aid from the New Hampshire Heart Association.

Read at the Thirteenth Annual Meeting of the Society for Vascular Surgery, Atlantic City, N. J., June 7, 1959.

*Professor of Clinical Surgery, Boston University School of Medicine; Visiting Surgeon, Massachusetts Memorial Hospitals, Boston; Chief, Division of Surgery, Huggins Hospital, Wolfeboro, N. H.

**Graduate Student Candidate for Master of Science Degree, Massachusetts Institute of Technology, Boston; now, Associate Professor of Mechanical Engineering, University of Durham, N. H.

The Korotkow full cuff method,[1] which every clinician uses in his blood pressure cuff, is well understood and more widely used than all other methods of blood pressure combined. The theory of a partial cuff was studied and tested in application. Instead of total imperforation as with the conventional cuff, the partial cuff only affects the area immediately adjacent to the artery under study. This system is relatively simple in theory and application and will yield accuracies equal to the sphygmomanometer.

Even though some of these indirect techniques may have some potentiality, all except the full cuff are complicated to use. It was felt that if some measurable parameter of the cardiovascular system could be found to vary in accordance with arterial pressure, then possibly that variable could be measured with greater ease but equal accuracy to continuous blood pressure recording through an intra-arterial cannula. If one knew the functional relationship between such a parameter and arterial pressures, then a direct correlation could be developed. Indeed, such a parameter exists and will now be described.

The majority of the tissues within the body are long chain polymers, or protein macromolecules. Since these protein molecules are elastomeric, one can predict[4, 5] the behavior of arterial sections under pressure. Analytically and experimentally, arterial volume varies in a near-linear manner with pressure. There is a straight line relationship over a large range of pressures. The functional relationships will vary from patient to patient, especially as influenced by age. Thus, if one measures arterial volume and correlates these volume measurements with pressure, the resulting relationship is one of close correlation, even under dynamic conditions. Since the relationship is quasilinear, definition of the slope of the curve and of one point on the curve establishes the correlation for any given patient. Mathematically, the functional relationship between pressure and volume can be given by the formula pressure (P) equals slope (M) times volume (V) plus a constant (b). The constant is defined by the point on the curve, and herein must be zero.

A calibration procedure was developed which determines these two mathematical characteristics for each case:

$$\text{Pressure Change } (\Delta P) = \text{Density } (p) \times \text{Level Change } (h)$$

(Based on blood specific weight of 1.057)

$$\Delta P = 0.74 \text{ mm. Hg per centimeter of Level Change}$$

Thus 33.8 cm. of level change induces a mean blood pressure variation of 25 mm. Hg.

A priori, a pressure change is induced within an artery by elevation of the arterial section with respect to the heart; that is, by displacing the arm vertically by 33.8 cm. a pressure change of 25 mm. Hg will be induced (Fig. 1). Experimentally, this has been verified to within 2 to 5 per cent. Furthermore, the pressure in a downstream section of an occluded artery will be atmospheric pressure (Fig. 2). Therefore, a method exists for establishing the slope of the curve and of one point on the curve, which is the zero point (Fig. 3). Thus, a measurement of arterial volume can be transcribed into a statement of blood pressure.

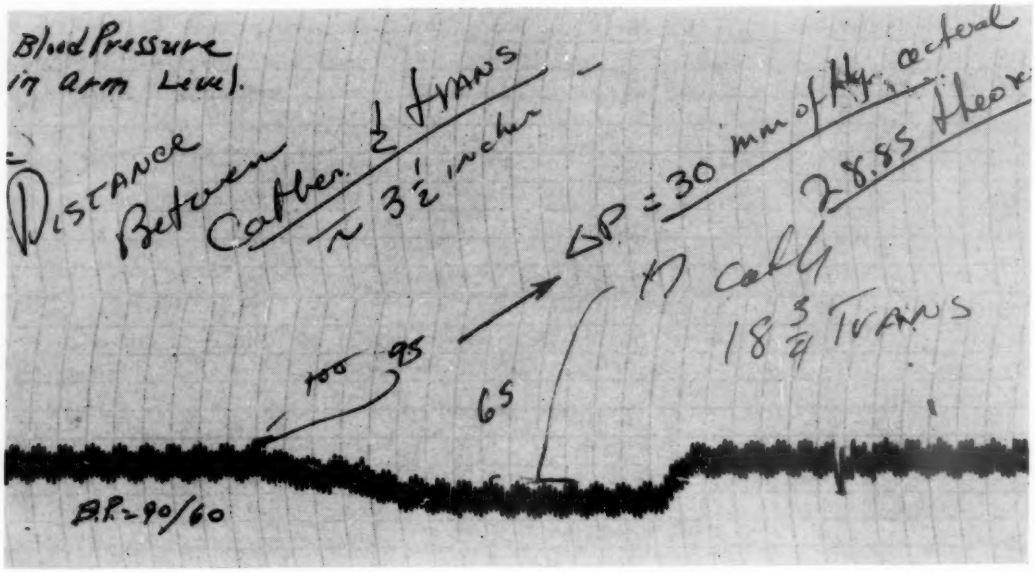


Fig. 1.—Tracing indicating fall of intra-arterial blood pressure by 25 mm. Hg for each 33.8 cm. by which arterial section is elevated with respect to heart level.

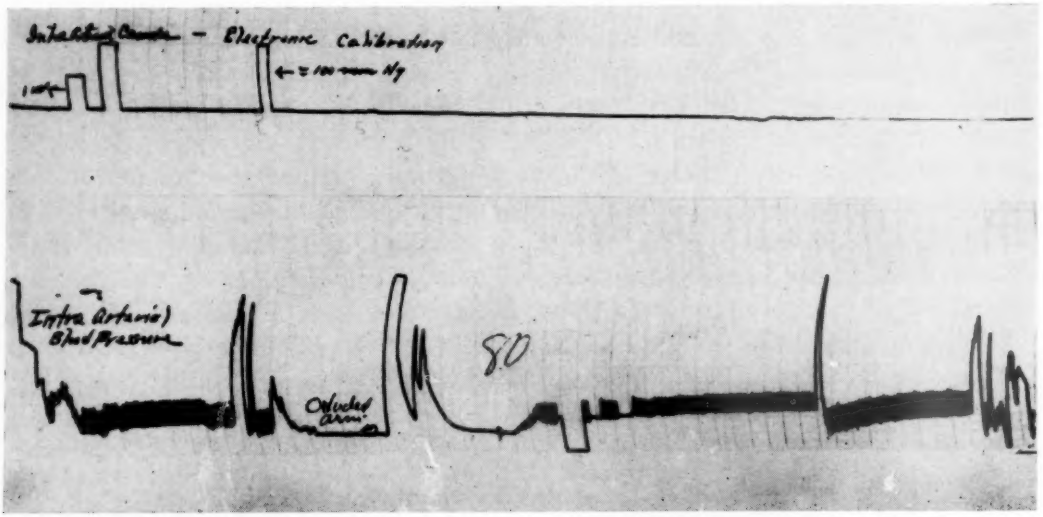


Fig. 2.—Actual tracing of an occluded arterial section as recorded by cannula. Note surge effect produced by mechanical blockage as pressure is being applied which shuts arterial lumen and reduces pressure to zero.

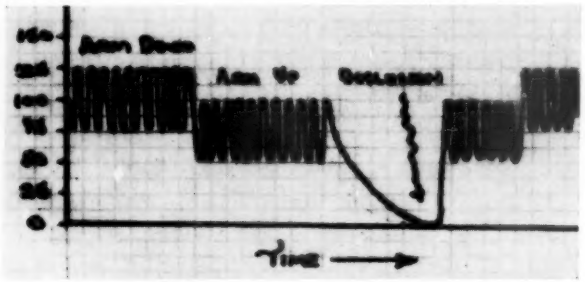


Fig. 3.—Graphic presentation of calibration procedure. During hydrostatic elevation the recording equipment is calibrated to give proper pulse pressure level. During occlusion, the mean pressure level is adjusted to read zero.

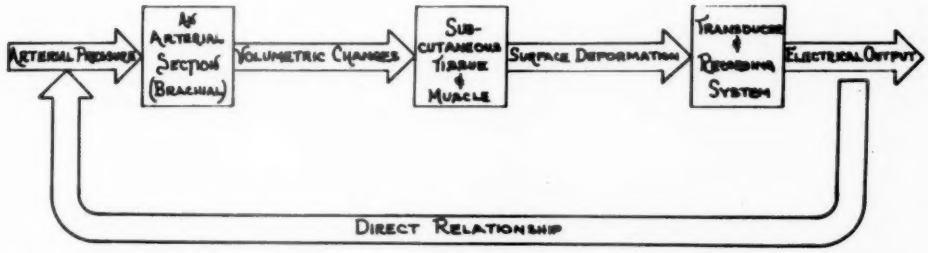


Fig. 4.—Pressure variable parameter blood pressure recording system. Schematic representation of the theoretical model of the entire system. Since there are quasilinear relationships between each parameter, the input and the output are directly related.

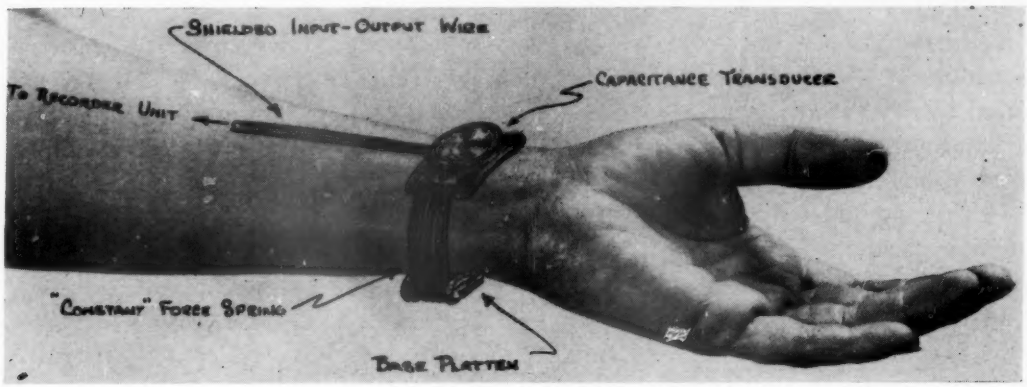


Fig. 5.—Sketch of miniaturized transducer in position on forearm.

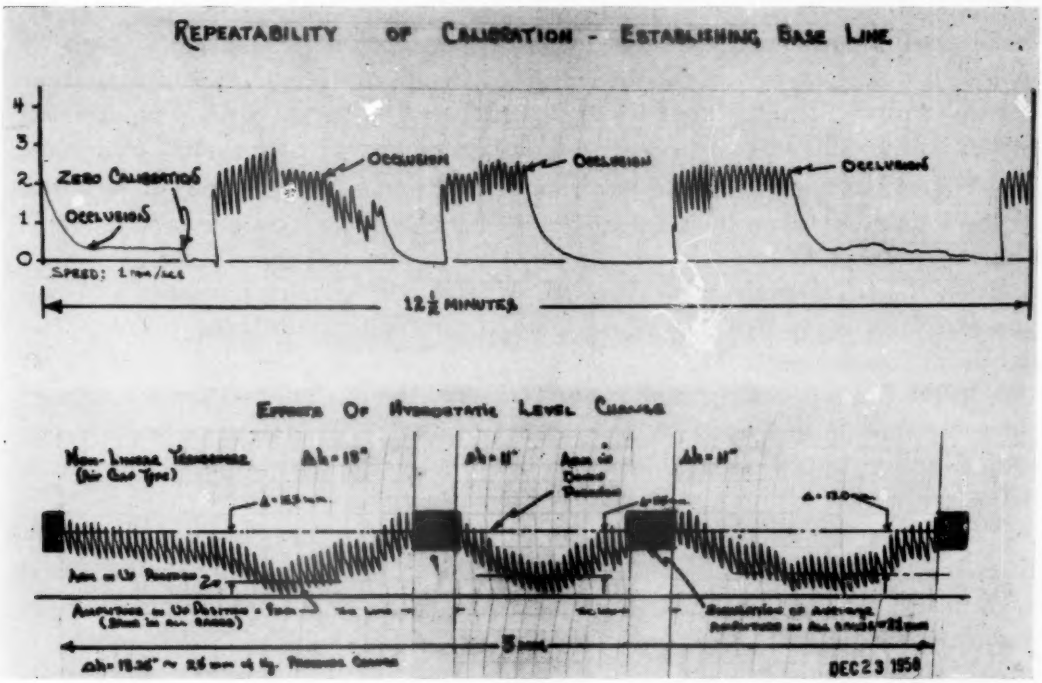


Fig. 6.—Repeatability of calibration data. *Top*, Base line or zero pressure. *Bottom*, Pressure response to change in hydrostatic level.

From analysis of a theoretical model,[2] it seems apparent that the surface deformations of the skin superficial to an artery are linearly related to intraluminal pressure (Fig. 4). In block diagram form, the intra-arterial pressure is directly related to an output signal, provided a suitable transducer can be developed.

After considering various implications of this concept, a capacitance transducer was designed for placement over an artery. The deformations of the skin surface must be measured accurately. Theoretically, one can measure either the volumetric changes or the actual diametral changes of the artery. The latter is much less complex and consequently was adapted to the system.

If there exist two electrically conductive plates separated by a suitable diaelectric-like air, they form a parallel plate capacitor. By suitable electronic monitoring, the distance changes between these two plates can be directly related to an output potential. In our transducer, one of these plates is made of a conductive flexible material and is placed over the arterial section. This plastic membrane, which is vacuum-coated with aluminum, has a low modulus of elasticity and will pulsate in accordance with the pulsatile variations of the surface tissue. The other plate of the capacitor is fixed in reference to the membrane, thereby forming a variable air gap capacitor whose capacitive variance is related to surface distentions of the tissue. As previously mentioned, this capacitive variation can be directly related to an electrical potential which can drive suitable recording equipment.

Based on this theoretical approach and experimental transducer, a prototype system was built and evaluated. The actual transducer has taken many forms during development, and the unit currently under evaluation is depicted in Fig. 5. Earlier, investigational programs were undertaken with more cumbersome units. The data now being reported were obtained with some of these earlier units, and indicate the results of preliminary developmental programs.

The calibration procedure has been tested under different conditions (Fig. 6), and some of the results from an early nonlinear system are shown. This system provided valuable data in spite of the nonlinearities. In the arm-down position the blood pressure is elevated, while in the arm-up position the blood pressure level is lowered. During occlusion, the pressure reduces to zero. The nonlinearities are electronic in origin, and these manifest themselves as an alteration in the pulse pressure amplitude. Intra-arterial cannulation data show that the amplitude is unaltered by this level variation. With a linearized system, under highly controlled conditions, the procedure has been accurate to within a few per cent.

The real test of the system is its response under clinical operative conditions. The output characteristics were compared by direct superposition and correlation (Fig. 7) on a double channel oscilloscope. It is apparent that the system has some inherent errors. One notes the phase shift in this case of approximately 35 degrees, and the damping effects due to elastomeric absorption. Under controlled conditions, the accuracy has been of the order of 3 to 8 per cent (Fig. 8). These are obviously stable conditions. Under variable pressures, the data correlation has been more of the order of 5 to 15 per cent (Figs.

9 and 10). These recordings are from clinical cases undergoing operative procedures and indicate results that are possible from a clinical research point of view.

Even though the correlation frequently is very good, repeatability and dependability remain poor at the present time.

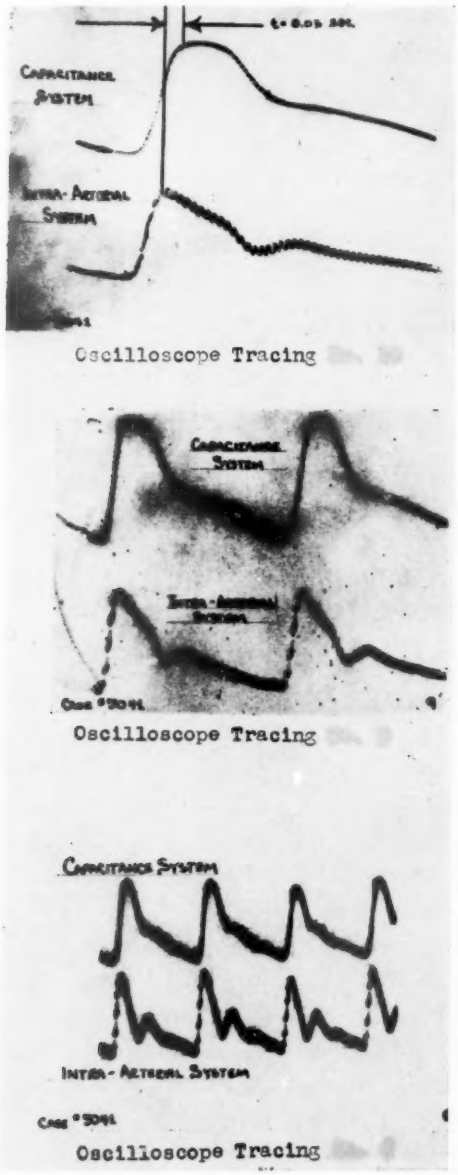


Fig. 7.—Comparison of output characteristics (blood pressure) readings as obtained from capacitance transducer and from intra-arterial cannula and transducer. Note the phase shift and damping effects.

There are time-dependent errors which limit the present application of the system. The maximum time for retention of calibration has been 90 minutes, but that was an exceptional case. Retention of calibration for 15 to 25 minutes is at present all that is possible. These time-dependent errors are not yet fully understood.

Transducer position has much influence on accuracy of the unit. In fact, many recordings have been invalid because of poor positional adjustment (Fig. 11). By altering the position of the transducer with respect to the surface, the characteristics are completely changed. The four tracings of Fig. 13 were obtained after what was intended to be identical positional adjustment procedure. In set I, the characteristics are accurate, but sets II and III are invalidated by collateral fact. Trial and error adjustment led to set IV, again identical to set I.

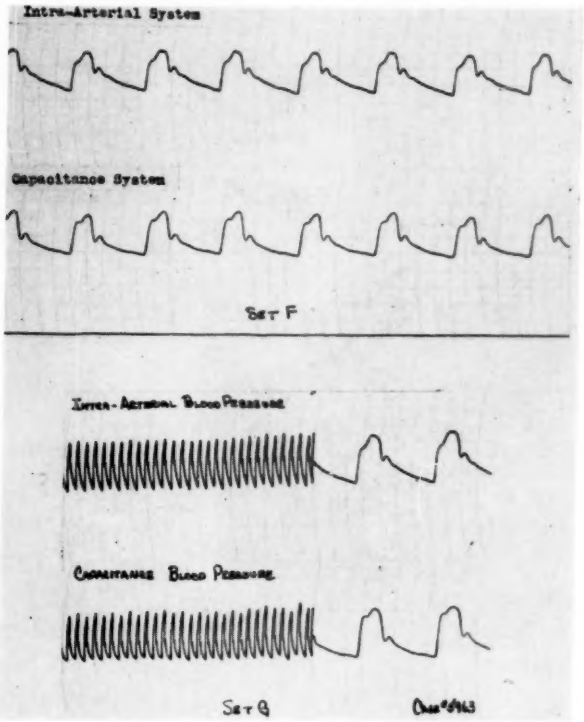


Fig. 8.—Demonstration of near identity in readings from intra-arterial and capacitance systems of recording, under stable conditions.

Integrally related to the positional problem is that of muscular relaxation. Some cases, particularly the nonanesthetized, show marked effects from muscular relaxation. This has the effect of changing the transfer characteristics of the system, and the time-dependent errors are largely of this origin. Most anesthetized cases do not show this effect, and the system is much more accurate under these conditions. Muscular contraction can be destructively disturbing to accuracy, but features have been designed and incorporated into the transducer mounting which permits actual muscular movements to be filtered out of the system. Thus, it is not movement that alters the characteristics, but relaxation of the mechanical structure of the muscle fibers and tissue.

Vasoactive drugs, whether constrictive or dilatory, have appeared to detract from accuracy, but in ways which have not yet been completely assayed. Plethysmographic similarities have not been recognized. A newly designed miniaturized transducer is expected to yield more information about the actual

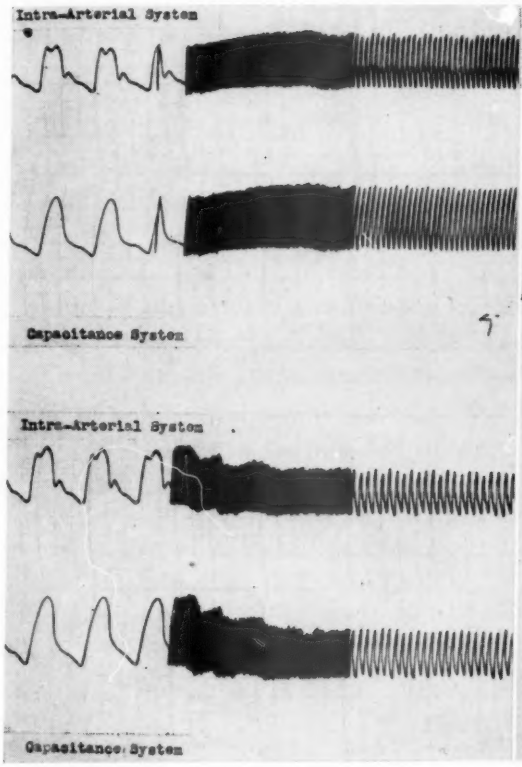


Fig. 9.

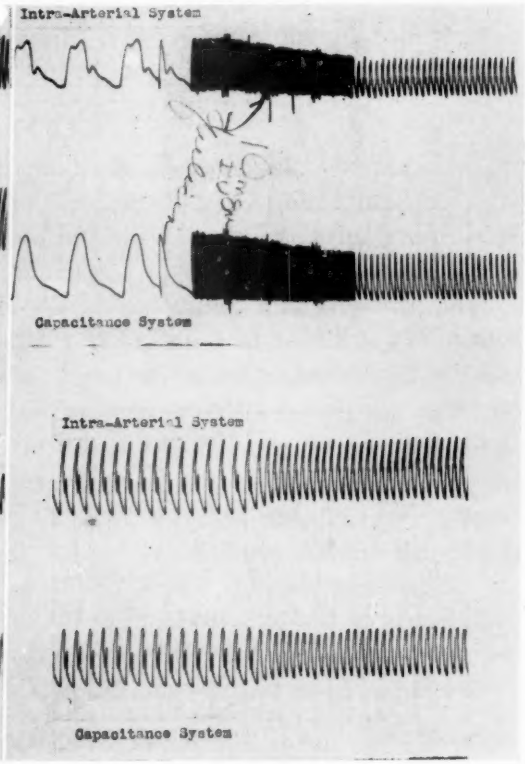


Fig. 10.

Fig. 9.—Response under clinical conditions that are variable, with pulse pressure errors of the order of 15 per cent and with considerable damping in the wave form.

Fig. 10.—Another clinical example of the two recording systems when clinical condition is changing rapidly. Note the damping effects in wave form.

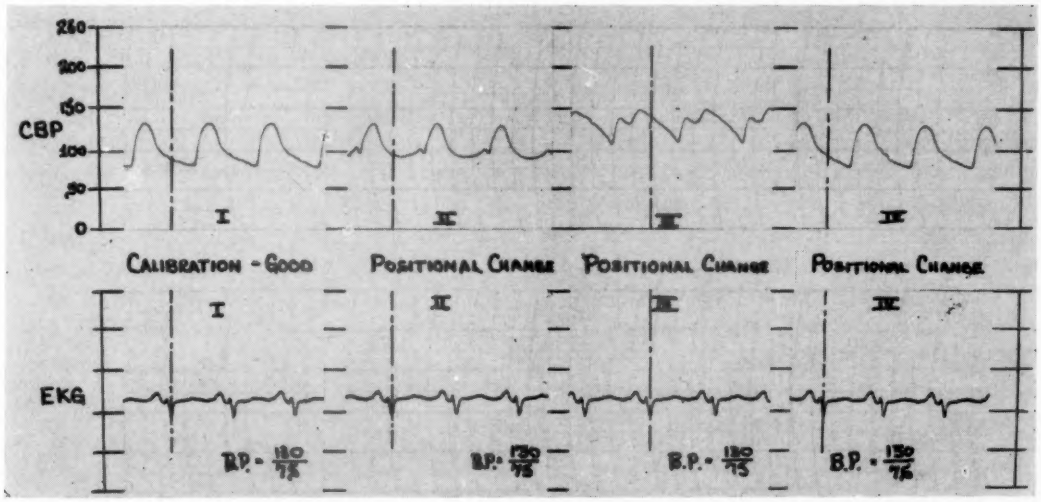


Fig. 11.—Serial tracings indicating critical nature of interface relationship between skin and transducer surfaces.

potentialities of this system. As indicated, many problems must yet be overcome before anyone can give this system serious consideration for wide clinical application.

SUMMARY

Capacitance measurement has been added to sphygmomanometry and intra-arterial cannulation as a third practical method of recording blood pressure. What peripheral resistance is to luminal pressure by mechanical measurement, "volumetric" displacement is to capacitance by electrical measurement. Since luminal pressure and volumetric expansion are related at a given moment, under conditions of either mechanical or electrical measurement, a reading of capacitance is reproducibly translatable into a statement of pressure.

The advantage is that readings can be made continuously, painlessly, and indefinitely with convenience equal to that of the cuff and stethoscope, and with accuracy approaching that of the intra-arterial cannula. The disadvantages are initial cost and the disturbing effects of small positional variation, or changes in muscle tension.

REFERENCES

1. Arynchin, N. I., and Zonkevich, I. S.: Half Century of Application and Further Development of Korotkow's (Russian spelling—Krotkov's) Sound Method for the Determination of Human Blood Pressure, Fiziol, Zhur. SSSR. **43**: 92-95, 1957. Referat Zhur. Biol. 1957, No. 70741. (trans. by U. S. Joint Publ. Res. Serv.)
2. Corell, Robert W.: Theoretical Analysis and Preliminary Development of an Indirect Blood Pressure Recording System. Thesis submitted in partial fulfillment of requirements for Master of Science Degree, Mass. Institute of Technology, Cambridge, Mass., May, 1959.
3. Hallock, P., and Benson, I. C.: Studies on Elastic Properties of Human Isolated Aorta, J. Clinical Invest. **16**: 595, 1937.
4. King, A. L.: Law of Elasticity for an Ideal Elastomer, Am. J. Physiol. **14**: 28, 1946.
5. King, A. L.: Pressure-Volume Relations for Cylindrical Tubes With Elastomeric Walls, J. Appl. Physiol. **17**: 501, 1946.
6. King, A. L.: Velocities of Pulse Waves in Large Arteries, J. Appl. Physiol. **18**: 595, 1947.

Discussion

DR. ROBERT S. SHAW (Boston, Mass.).—This sort of problem is not too pertinent in the hospital but is of considerable interest to the Air Force because of the need to monitor blood pressure in subjects traveling at great speeds, all alone, many hundreds of miles away from the physician. Under these circumstances an intra-arterial needle may represent a danger as well as uncertainty.

There are a lot of problems that I do not think are quite yet solved by this, but I certainly would congratulate the authors for their effort and what has been accomplished.

The two particular things that are troublesome in this sort of approach are the variation in pressure-volume relationships in an artery with variations in vasomotor tone, and also the variations in tissue volume that occur with engorgement of tissues as a result of variations in venous pressure, but I think this is a fine piece of work and quite a new idea.

DR. ADAMS (closing).—Physiologic variations as they occur in clinical patients in the operating room can now be electronically recorded or magnified in ways that permit anticipation, and frequently control, of many changes in conditions that formerly were hard to detect until far advanced. Reliable managing of vasomotor tone during periods of stress should have significant guidance value. It is hoped that continuous cuffless noncannula blood pressure recordings may be feasible through a device such as has been described here.

THE LAWS OF FLUID FLOW AND ARTERIAL GRAFTING

D. Emerick Szilagyi, M.D., John G. Whitcomb, M.D., Walter Schenker, M.D., and Peter Waibel, M.D., Detroit, Mich.

(From the Department of General Surgery, Henry Ford Hospital)

As long as the surgeon was restricted to the use of homografts and simple replacement techniques in reconstructive surgery of the arteries, the problem of interfering with normal hemodynamics did not loom large. The physiologically significant properties of the new arterial pathway were a good deal like those seen, under normal conditions, in the reconstructed segment. The more recently introduced angioplastic techniques, however, often boldly modify many of the factors that determine the flow characteristics of the circulating blood. The new vascular channels may differ significantly from the host arteries with respect to length, diameter, configuration, and surface texture. These deviations have been particularly evident since the advent of plastic arterial prostheses that present many types of manufacture and a wide variety of dimensions.

Although these physiologic artefacts created by the surgeon must have bearing on blood flow and, therefore, on clinical results, quantitative observations on this relationship have not been reported. Even in the experimental area, published data pertinent to these problems have been scanty and have been obtained from studies on artificial models.[7, 10] Indeed, owing to the lack of reliable metering devices for the study of the volume flow of blood applicable to operating room studies, clinical observations of this type are extremely difficult if not impossible to gather. The experimental investigation of these factors likewise raises special problems, such as the reproduction in the experimental animals of the anatomic and physiologic conditions of the human vascular system and the maintenance of comparable hemodynamic states in the experimental animals and their controls.

In the studies to be reported a preparation was employed that utilizes the experimental animal as its own control. In this preparation, one iliac artery is made ready for the experimental flow study while the other serves as control. The various arterial and prosthetic replacements are implanted in the experimental side, and the volume flow of blood is determined by simultaneous paired measurements at the distal ends of both iliac arteries. Under these conditions the experimental factors affecting blood flow are bilaterally identical except for the planned variable introduced by the arterial substitute. In what follows we wish to report a description of this method as well as the results obtained by its use.

The experimental work was supported in part by a grant from the Michigan Heart Association.

Read at the Thirteenth Annual Meeting of the Society for Vascular Surgery, Atlantic City, N. J., June 7, 1959.

METHOD

The factors with bearing on hemodynamic behavior studied were: (1) the type of anastomosis, that is, end-to-end compared to various types of end-to-side anastomoses; (2) bypass grafting as compared to direct replacement; (3) the diameter of the implant; and (4) the luminal surface characteristics of the implant. In all four groups of experiments the basic plan was the same: the vascular substitute to be studied was inserted on the experimental side in a predetermined manner while the other side was either left intact or, occasionally, subjected to a control operation, and the rate of volume flow of blood was determined simultaneously on both sides, with every effort being made to assure bilaterally identical physiologic conditions except for the factor investigated.

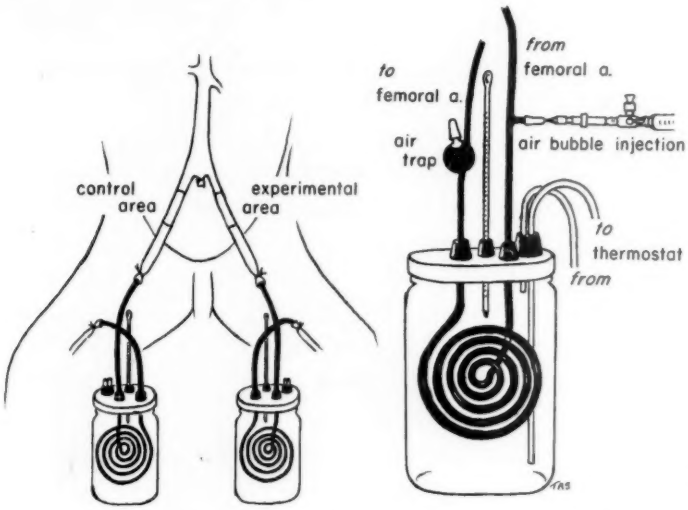


Fig. 1.—Diagram of measurement of blood flow with bubble flowmeter.

In dogs weighing 30 to 70 pounds the aortic bifurcation and iliac arterial systems were exposed through a long midline incision under Nembutal anesthesia. After both iliac arteries had been dissected free from their beds, on the experimental side the technical procedure to be studied was carried out. For the measurement of blood flow the bubble flow principle was used as described by Dumke and Schmidt (Fig. 1).[2, 6] Both the control and the segment of the experimental iliac artery distal to the area of surgical interference were connected by means of appropriate polyethylene tubing to separate bubble flow meters (in a constant temperature bath), 0.2 ml. of air was injected into the afferent lead of each meter, and the speed of the bubble propelled by the flowing blood through a glass coil of known capacity was accurately timed; a simple calculation gave the amount of blood traversing the meter during unit time. The injected air bubble was trapped on the efferent side of the meter before the blood was shunted back into the distal iliac arterial trunk. By allowing the blood to return to the distal arterial tree after traversing the flowmeter, the peripheral resistance was left essentially undisturbed and the effects of an arteriovenous fistula—that would have been created had the arterial blood been returned to the iliac vein—were avoided. During the determination of the blood flow through the experimental preparation the systemic blood pressure as well as the pulse rate were continuously recorded through a carotid cannula. Measures were taken, when needed, to keep the hemodynamic conditions during each experiment as constant as practicable. During arterial occlusion necessary for the construction of the various anastomoses the distal arterial beds were heparinized, and systemic heparinization was practiced during the actual meter runs (by the intravenous administration of 2 mg. of heparin per kilogram of body weight). The glass surfaces of the instruments in contact with blood had been coated with silicone. In making the vascular anastomoses the conventional clinical instruments and techniques were employed.

For each experiment 2 to 10 animal preparations were made. Every recorded result was the calculated mean value of at least six paired determinations on the same preparation. If the serial readings in a given experiment deviated by more than 15 per cent the experiment was discarded. As a further control, at the completion of each experiment, blood flow was measured by a 15-second collection of blood from the distal ends of the iliac trunks. Thereafter, the anastomoses were taken down and inspected for the presence of stenosis or thrombotic occlusion; the presence of such artefacts invalidated the experiment.

The values of the blood flow on the experimental side were expressed as percentages of the flow determined simultaneously on the control side.

The subsidiary problems in each of the four groups of experiments mentioned above were as follows:

Group 1. Type of Anastomosis.—(A) Comparison of end-to-end anastomosis to intact control (Fig. 2). (B) Comparison of end-to-side anastomosis (end of graft to side of host) to end-to-side anastomosis of the control with variations in the angle of anastomosis on the experimental side (Fig. 3). (C) Comparison of side-to-end anastomosis of the control with variations both in the technique and in the angle of the anastomosis on the experimental side, a simple slit anastomosis being tested against a bell-mouthed anastomosis (Fig. 4). It should be noted that in creating a bell-mouthed anastomosis a brim of tissue is excised from the margins of the slit in the wall of the recipient artery; the favorable elastic deformation of the host artery widens the opening and reduces or eliminates the sharp edges of the orifice.

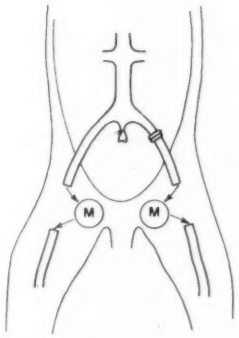


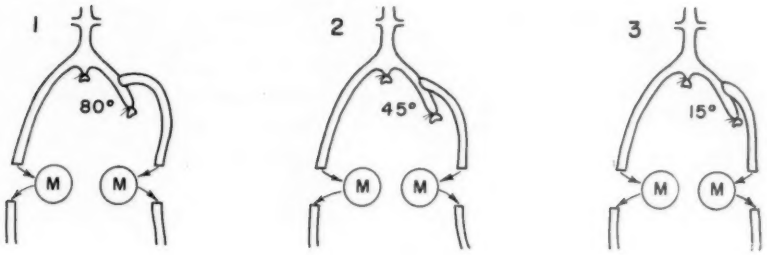
Fig. 2.—Comparison of flow through a single end-to-end anastomosis with flow through intact artery. (In this figure as well as in the following figures the symbol *M* in a circle denotes the location of the flowmeter.)

Group 2. Bypass Grafting (Fig. 5).—(A) Comparison of direct replacement to intact control. (B) Comparison of bypass graft to intact control. (C) Comparison of bypass graft to direct segmental replacement. (D) Comparison of bypass graft inserted with a bell-mouthed anastomotic opening to direct segmental replacement as control.

Group 3. The Effect of Variation in Graft Diameter (Fig. 6).—(A) The effect of an increase in diameter of 60 per cent over the control. (B) The effect of an increase in diameter of 100 per cent over the control. (C) The effect of an increase in diameter of 140 per cent over the control. (D) The effect of an increase in diameter of 170 per cent over the control. In all these experiments the experimental implant was a graft of human origin and the anastomoses were end-to-side.

Group 4. The Effect of Luminal Surface Characteristics of Prosthesis (Fig. 10).—(A) Comparison between crimped braided nylon prosthesis and human femoral arterial graft. (B) Comparison between crimped knitted Dacron prosthesis and human femoral arterial graft. (C) Comparison between crimped woven Dacron prosthesis and human femoral arterial graft. (D) Comparison between elastic Dacron prosthesis and human femoral arterial graft. The principal parameter here was the effect of corrugation and ridging of the inner surface of the prostheses caused by crimping. All the crimped prostheses (A, B, and C), in addition, showed some degree of minor surface roughness. The latter effect was also present in the uncrimped prosthesis (D).

Fig. 3.



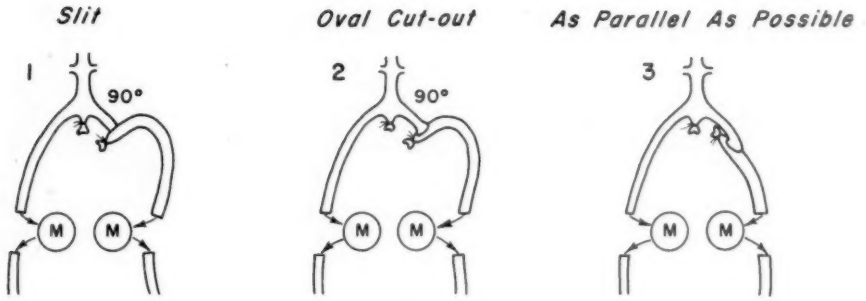


Fig. 4.

Fig. 3.—Variations of technical procedure for the study of end-to-side anastomosis.
Fig. 4.—Variations of technical procedure for the study of side-to-end anastomosis.

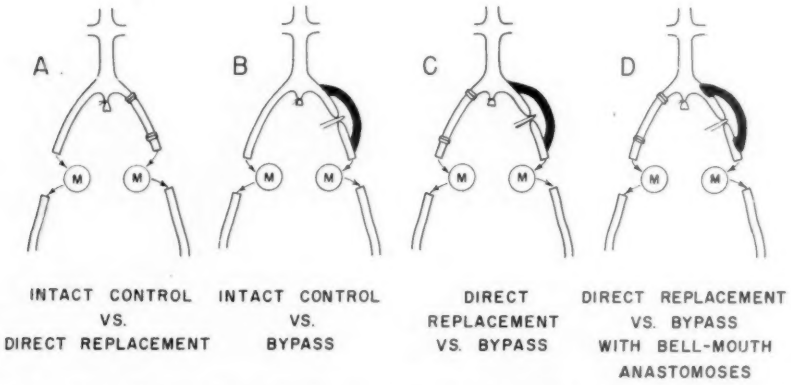


Fig. 5.—Variations of technical procedure for the study of bypass grafting.

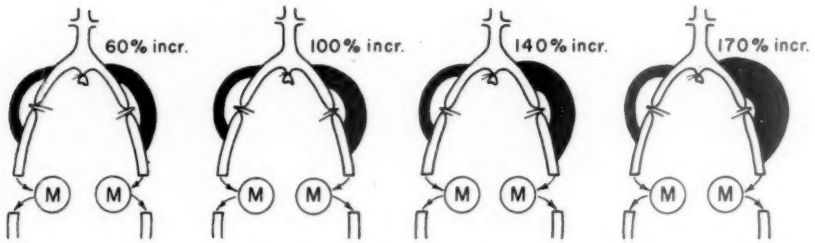


Fig. 6.—Study of graft size.

RESULTS

The findings of these experiments are listed in summary form in Tables I through IV and Figs. 7 through 10.

The results in subgroup A of the experiments under Group 1 (Table I, Fig. 7), undertaken to explore the effects of the type of anastomosis on volume flow of blood, showed that a properly performed end-to-end anastomosis did not appreciably interfere with blood flow. The remaining experiments in this group aimed at gathering information about the possible influence of two technical details

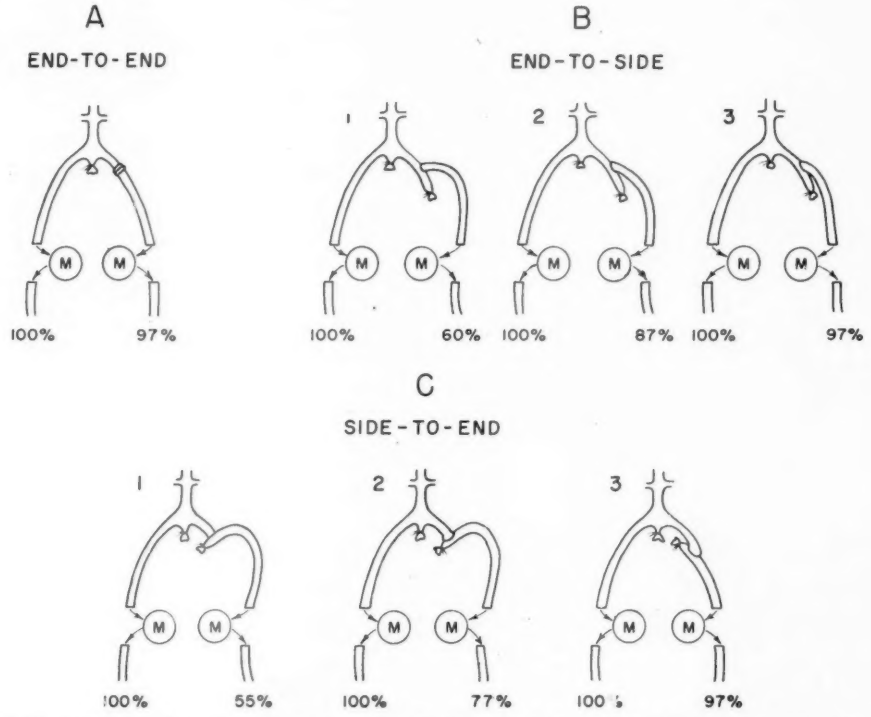


Fig. 7.—The results of flow measurements in the study of single anastomoses of various types of construction. In this as in Figs. 8, 9, and 10 the volume flow on the experimental side is expressed as the percentage of the flow on the intact side.

TABLE I. EFFECT OF TYPE OF ANASTOMOSIS ON BLOOD FLOW
(Group 1)

SUB-GROUP	TYPE OF ANASTOMOSIS		NUMBER OF EXPERIMENTS	BLOOD FLOW ON CONTROL SIDE (ML./MIN.)		BLOOD FLOW ON EXPERIMENTAL SIDE (ML./MIN.)		FLOW ON EXPERIMENTAL SIDE EXPRESSED AS PER CENT OF FLOW ON CONTROL SIDE	
	CONTROL SIDE	EXPERIMENTAL SIDE		RANGE	MEAN	RANGE	MEAN	RANGE	MEAN
A	Intact vessel	End-to-end	2	28.7-53.6	37.4	28.0-56.2	36.7	96- 99%	97%
B	End-to-end	End-to-side with varying angles	7	17.3-73.8	—	16.8-79.8	—	60-100%	—
C	End-to-end	Side-to-end	3	43.2-78.9	—	35.5-46.1	—	55- 98%	—

End-to-side denotes an anastomosis of the end of the graft to the side of the host artery; side-to-end, the opposite.

concerned with the performance of end-to-side anastomoses: the angle at which the arteries forming the anastomosis were approximated, and the size and shape of the anastomotic opening in the recipient artery. One fact stood out rather conspicuously, namely, that the more acute (or smaller) the angle between the component arteries, the more efficient was the anastomosis. A longer anastomotic opening with the removal of a brim of tissue from the margins of the slit in the wall of the recipient artery (so-called bell-mouthed orifice) likewise increased the efficiency of the anastomosis (Figs. 7, *C-2* and *C-3*). It should be noted in this connection that, in regard to the size of the anastomotic opening in end-to-side anastomosis, it is closely related to the angle of union of the component arteries, that is to say, the longer opening will require a sharper angle on the slant of the cut end of the graft which, in turn, results in narrower angle of union.

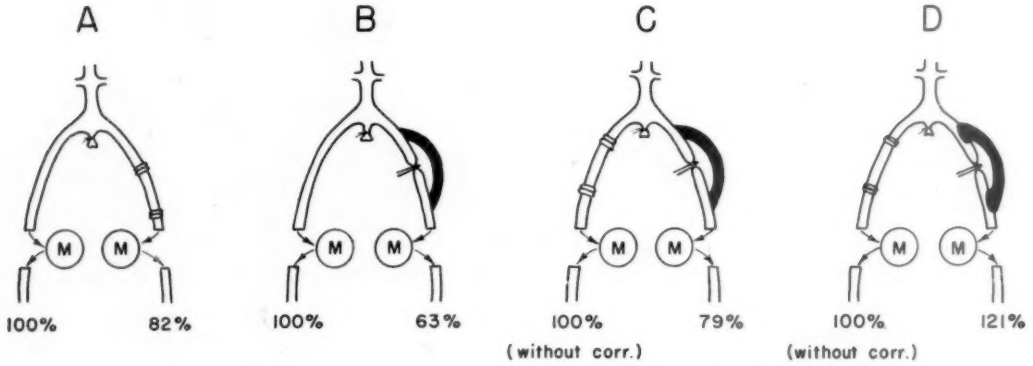


Fig. 8.—The results of flow measurements in the study of bypass grafting. In *C* and *D*, the flow on the experimental side is compared *not* with an intact artery but rather with the flow through a direct replacement graft; since the flow through the replacement graft is less than through an intact artery (by about 18 per cent), in order to obtain the flow through the experimental side in terms of the flow through the intact artery, a correction factor should be applied. After this correction the flows in *C* and *D* should become about 73 per cent and 100 per cent.

In the Group 2 experiments for the study of the efficiency of bypass grafting (Table II, Fig. 8) in subgroup A, the direct replacement was constructed with end-to-end technique both proximally and distally. In subgroups B and C the

TABLE II. BLOOD FLOW THROUGH BYPASS GRAFTS
(Group 2)

SUB-GROUP	OPERATIVE TECHNIQUE		NUMBER OF EXPERI-MENTS	BLOOD FLOW ON CONTROL SIDE (ML./MIN.)		BLOOD FLOW ON EXPERIMENTAL SIDE (ML./MIN.)		FLOW IN IMPLANT EXPRESSED AS PER CENT OF FLOW IN CONTROL	
	CONTROL	IMPLANT		RANGE	MEAN	RANGE	MEAN	RANGE	MEAN
A	Intact vessel	Direct segmental replacement	3	22.6-59.5	38.4	20.8-40.7	31.5	68.5-92.0%	82%
B	Intact vessel	Bypass	3	32.7-51.4	43.1	22.2-40.8	27.0	50.0-79.0%	63%
C	Direct segmental replacement	Bypass (slit anastomosis)	4	19.8-72.0	44.3	13.4-55.2	36.5	63.0-94.0%	79%
D	Direct segmental replacement	Bypass (special anastomosis)	3	25.9-57.0	42.2	30.0-75.4	52.0	106-140%	121%

bypasses were constructed in an identical manner, using end-to-side slit anastomoses; in these preparations the variables were on the control side, which in subgroup B was left intact and in subgroup C had a segmental replacement with proximal and distal end-to-end anastomoses. In subgroup A only a moderate reduction of flow occurred on the experimental side which must have been the result of the summation of the slight reduction of flow at each of the two end-to-end anastomoses. The volume flow through the bypass was significantly reduced in both subgroup B and subgroup C. The findings in subgroup D were of interest. In these experiments the anastomoses between the grafts and the host artery were performed at a very acute angle and with bell-mouthed openings, and a significant improvement in flow was observed in the bypass. It will be noted that in order to obtain the true rate of flow through the bypasses in subgroups C and D a correction factor must be used, since in these experiments the bypasses are compared to segmental replacements which in themselves cause a reduction of flow; the correction factor is about minus 20. It should be made clear that the segment of artery bypassed by the graft was in all cases of Group 2 completely occluded.

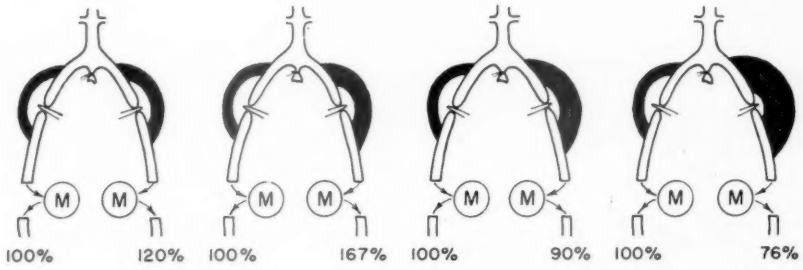


Fig. 9.—The results of flow measurements in the study of the variations of graft diameter.

The experiments in Group 3 dealing with the effect of the variation of graft diameter (Table III, Fig. 9) demonstrated that an increase in diameter up to twice the diameter of the control brings about an augmentation of volume flow which, however, is not a linear change. With an increase of 60 per cent in the diameter an average increase in flow of 20 per cent was found, while an increase in diameter of 100 per cent produced an increase of 67 per cent. Increasing the diameter by a factor of 2.4 and 2.7 resulted in a decrease of volume flow.

TABLE III. EFFECT OF VARIATION IN GRAFT DIAMETER ON BLOOD VOLUME FLOW
(Group 3)

SUB-GROUP	CONTROL TO GRAFT RATIO	NUMBER OF EXPERIMENTS	VOLUME FLOW IN CONTROL (ML./MIN.)		VOLUME FLOW IN GRAFT (ML./MIN.)		VOLUME FLOW IN GRAFT AS PER CENT OF FLOW IN CONTROL	
			RANGE	MEAN	RANGE	MEAN	RANGE	MEAN
A	1:1.6	10	12.2-24.9	18.4	13.8-34.6	22.3	112-139	120
B	1:2.0	10	13.2-24.7	23.4	21.2-41.6	32.7	149-187	167
C	1:2.4	6	17.0-52.1	25.5	14.9-48.4	23.2	81-104	90
D	1:2.7	4	18.3-27.3	22.0	14.5-20.4	16.4	72- 79	76

The grafts studied were fresh human femoral arteries. The length of the grafts was uniform (5 cm.); both proximal and distal anastomoses were end-to-side.

The experiments in Group 4 (Table IV, Fig. 10), planned to compare the hemodynamic properties of a number of vascular plastic prostheses of various types of construction, disclosed that flow through the prosthesis with the least surface irregularities (D) was the most efficient.

TABLE IV. EFFECT OF LUMINAL SURFACE CHARACTERISTICS OF PROSTHESES ON BLOOD VOLUME FLOW
(Group 4)

SUB-GROUP	TYPE OF PROSTHESIS	NUMBER OF EXPERIMENTS	VOLUME FLOW IN CONTROL (ML./MIN.)		VOLUME FLOW IN PROSTHESIS (ML./MIN.)		VOLUME FLOW IN PROSTHESIS AS PER CENT OF FLOW IN CONTROL	
			RANGE	MEAN	RANGE	MEAN	RANGE	MEAN
A	Crimped braided nylon (Edwards-Tapp)	10	22.0-40.8	31.5	16.1-30.0	25.0	73-89	80
B	Crimped knitted Dacron	5	10.2-54.9	34.47	10.2-60.9	38.4	85-96	88
C	Crimped woven Dacron	5	16.9-48.2	27.32	18.8-54.4	32.7	75-88	85
D	Elastic woven Dacron	10	25.9-59.7	34.1	24.6-51.3	30.3	81-96	90

The controls were human femoral arterial grafts. All anastomoses were end-to-side. Length of all implants was 5 cm.; diameter of all implants 8 mm.

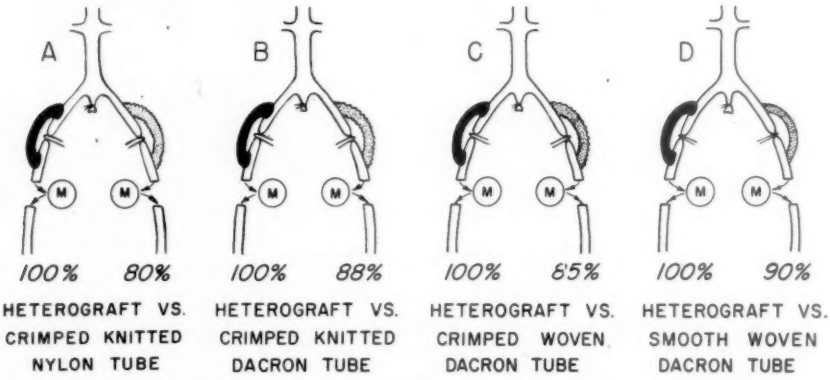


Fig. 10.—The results of flow measurements in the study of surface irregularities in the arterial substitute.

DISCUSSION

The Validity of the Animal Preparation as a Reproduction of a Human Arterial Graft.—An interpretation in precise physical terms of the hemodynamic phenomena observable in the human vascular system has not as yet been achieved. The difficulties in the way of such an interpretation at present seem formidable. The laws with which the physicist or engineer operates refer to hydrodynamic systems whose characteristics are constant or at least predictable. The solution of problems in such nonbiologic hydraulic systems is possible either by the manipulation of known mathematical formulas or by the establishment of new formulas as a result of precisely controlled experiments. Neither of these approaches is open to the human biologist. The mathematical formulas of fluid flow are applicable to the human vascular system only in a very coarse qualitative way, and the experimental study of even small segments of the animal vascular

system is open to countless errors. Indeed, with respect to the human circulatory system some of the primary facts needed for meaningful interpretation of experimental observations are lacking (such as the exact flow pattern prevalent in the various segments of the arterial tree, the nature of blood as a viscous or nonviscous fluid, the exact role of arterial elasticity, and others).

It is an obvious question, therefore, whether these limitations may not apply also to the experimental study of the problems of blood flow in arterial implants.

It seemed to us, a priori, that so long as the investigation of blood flow is limited *to an isolated segment of conduit* of stable dimensions, the conditions are essentially different and infinitely simpler than those present in the study of a complex biologic unit. It was reasoned that the critical requirements of the experiments under these conditions would not be the solution of the complexity of uncontrollable parameters that confront the student of the dynamics of the circulatory system as a whole, but rather, first, the need for the devising of an animal preparation that reproduces with reasonable faithfulness the simple physical properties of human arterial grafts and, second, the control of the systemic factors that even in an acute animal experiment constantly modify the flow of blood. The preparation described earlier fulfills the second requirement quite satisfactorily but is somewhat defective as a replica of its human prototype. The greatest shortcoming of the preparation is inherent in the limitations that the size and anatomy of the experimental animals impose on its dimensions. The arterial implants used in the experiments are good miniature reproductions of a human graft they are meant to represent, but their small size introduces hydrodynamic factors that must be kept in mind when the results of the experiments are interpreted. These will be discussed presently. The second requirement of the study, the control of extraneous variables, was more fully met. Since flow in the experimental side was always expressed as a ratio of the simultaneously observed flow of the intact side, the effect of irrelevant factors was virtually eliminated.

It should perhaps be pointed out that the results of flow measurement were in no way anticipated; that is to say, no preconceived assumptions were made in regard to observations that might have been predicted on the basis of hydrodynamic laws. The record of the measurements was submitted to consultants of the required knowledge of fluid mechanics only when the investigation had been completed.

In commenting on methodology, mention should be made of the reason for the choice of the bubble flowmeter as the measuring device. After rather exhaustive inquiry and a good deal of experimentation, we convinced ourselves that no ideal method of blood flow measurement is available, and among those instruments that were applicable to this study the bubble flowmeter had the attractive qualities of accuracy (a margin of error of 2 to 4 per cent at the rates of flow under consideration), ruggedness, and simplicity.

Some Elementary Physical Principles.—Before undertaking the interpretation of the results in terms of hydrodynamic principles, a brief statement of some elementary notions of physics will be in order. In a discussion of this type this statement can hardly be more than a list of nonmathematical definitions.

In physics an important distinction must be made between the terms *fluid* and *liquid*. A *liquid* is any physical body that is readily deformed by a tangential or shearing stress and remains deformed after the stress is removed; in contrast, a *solid* tends to regain its original shape after the deforming stress ceases. A *fluid* may be either a liquid or a gas, the principal difference being the degree of cohesion and compressibility of the molecular particles of each. A liquid is essentially incompressible and cohesive; a gas is highly compressible and expansile. Otherwise, however, the mechanical behavior of liquids and gases is alike and the laws governing their behavior are similar or identical.

A fluid may flow in one of two manners. When the flow is *laminar* (streamlined or viscous) the fluid moves in layers of infinitesimal thickness that slide one upon another; in the flow of this type, when it takes place in an enclosed space such as a pipe, the movement of the fluid can be conceived as the sum of the forward motion of concentric cylindrical layers, much like the movement of the parts of a collapsible telescope. The force that opposes this sliding or shearing motion of one layer upon another is viscosity which can be defined as a ratio of the shearing stress between the fluid layers to the rate of shear. Since the retardation in the movement of each layer of liquid by the viscous property of the layers adjacent to it is additive, the layers nearer the outer boundary in a pipe will flow slower, while the axial layer will have the highest velocity. Thus the contour of the flow velocity distribution of laminar flow has a characteristic parabolic shape. Laminar flow is the most efficient manner in which fluid moves since (disregarding surface friction) all the energy not dissipated by viscosity is expended for propelling the fluid forward. In the second type of flow, *turbulent flow*, the fluid particles, instead of moving in paths parallel with the direction of flow, follow irregular paths at various angles to the flow direction, thus creating eddies and dissipating part of the available energy as heat. Formulas describing the characteristics of laminar flow can be arrived at by mathematical analysis, while the description of the characteristics of turbulent flow must follow experimental observations and thus are often not predictable by abstract analysis alone.

Turbulence in the flow of a fluid will be brought about by a number of factors. Assuming a conduit of relatively small size, uniform diameter, and smooth (frictionless) surface, turbulence will appear when a certain ratio (called Reynolds' number) of inertial forces to viscous forces is exceeded. The inertial forces, an increase in which tends to cause turbulence, are density, the radius of the conduit, and the velocity of flow. The property of the fluid that enhances laminar flow is viscosity. In the ideal system just mentioned, turbulence will not occur until a Reynolds' number (N_R) of 1,000 to 2,000 is reached. Turbulence may occur at much lower N_R when the conduit wall is rough. It is assumed by most authors that in the normal human cardiovascular system the flow is seldom if ever turbulent. However, the objective proof of this assumption is lacking. Moreover, there is much clinical evidence that in human arterial systems with advanced intimal roughening and other arteriosclerotic changes the flow is frequently turbulent even in arteries of relatively small size.

The pressure/flow relationship governing laminar flow in narrow smooth tubes was formulated by Poiseuille whose law, phrased in words, states that the volume flow is directly proportional to the pressure drop across a given length of the conduit and to the fourth power of the radius of the conduit, and inversely proportional to the length of the conduit and to the internal friction of the fluid. Under stable conditions in a conduit of uniform dimensions the linear velocity and volume flow are proportional to the applied pressure. In considering Poiseuille's formula the importance of the effects on flow of small changes in the radius of the conduit is particularly noteworthy. One must also stress that this law is applicable only to the laminar flow of a homogeneous viscous fluid under constant pressure and in a frictionless rigid tube of uniform physical dimensions. The equation is only qualitatively applicable to the human vascular system which has highly variable dimensions, elastic walls, and constantly changing pressure head, and contains a nonviscous fluid.

In a stable system of moving fluid the total energy expended in performing work manifests itself in three forms: *the kinetic energy* (velocity head), *the potential energy* (static pressure, or pressure head) and *heat energy* (produced by friction). In the case of an ideal frictionless fluid, the sum of the kinetic and potential energies is constant, that is, an increase in one is always followed by a decrease in the other (Bernoulli's theorem). On the other hand, since at equal time intervals equal amounts of fluid pass through a given cross-sectional area of a conduit *("equation of continuity")*, at a narrowed segment the velocity of the fluid must increase, that is, the moving fluid will gain increased kinetic energy and as a result its potential energy or static pressure will decrease. At an enlarged segment of a conduit the converse will happen.

Irregularities at the boundary surface causes loss of flow owing to the eddies created by the impact of fluid against the projections. This *friction* effect will vary with the square of the velocity of flow. Surface irregularities will cause turbulence at a much lower flow velocity than will occur in a system with smooth walls and identical in other respects. The importance of a given surface irregularity in causing increased friction is expressed by its ratio to the total diameter of the conduit; that is, a surface irregularity of a given size will cause more friction in a tube of a smaller diameter than it would in a tube of larger diameter.

In the analysis of the hydrodynamic phenomena seen in experimental grafts —or in human arterial implants for that matter—the turbulence brought about by sudden alterations in the direction, velocity, and magnitude of the flowing stream is of particular interest. The presence of orifices and variations of tube dimensions in the path of flowing fluid will cause laminar flow suddenly to change into turbulent flow. This change, in turn, results in a decrease in the rate of flow owing to loss of energy, which is reflected in the slope of the hydraulic and energy gradient lines.

For an explanation of the meaning of the hydraulic and energy gradients reference is made to the idealized hydraulic system sketched in Fig. 11. The

parameters that describe the rate of flow through a horizontal, freely discharging pipe of uniform diameter in such a system are the total head *(H)*, pressure head *(h_p)*, velocity head *(h_v)*, and friction head *(h_f)*. (The term "head" in this context is essentially synonymous with "energy.") The total amount of potential energy (total head, H) available for propelling the fluid through the pipe is represented by the weight of the fluid in the reservoir lying above the midplane of the pipe. As the fluid enters the pipe a part of this energy is transformed into kinetic energy, supplying the velocity head (h_v or $v^2/2g$, v being the velocity of the stream in the pipe and g the gravity constant). The remaining potential energy is used to supply the pressure head (h_p or p/w since it is an equivalent height of liquid representing a pressure p in a fluid of specific weight w), and to provide for the loss of energy due to friction (friction head, h_f). The sudden drop in the pressure head in Fig. 11 between the entrance into the pipe and Fig. 11, *a* is mainly due to the change of potential energy into kinetic energy,

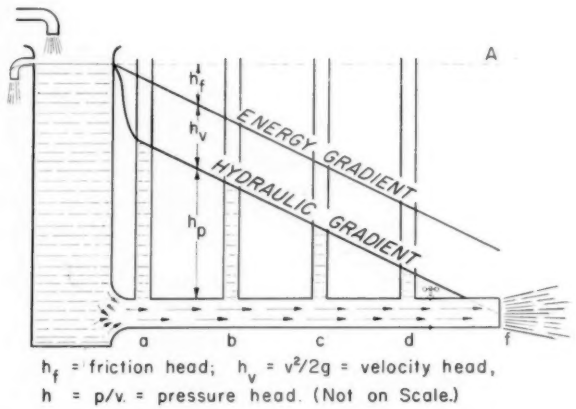


Fig. 11.—Energy gradient and hydraulic gradient lines in a stable hydraulic system. For explanation see text.

$v^2/2g$. It will be noted that in the system under consideration, the flow being steady, the magnitude of this velocity head remains unchanged. The further decrease in the pressure head at *b* and the subsequent points *c* and *d* is the result of friction loss. If one lays off a line connecting the points marking the local pressure heads in the standpipes (*a* to *d*) from the surface of the reservoir to the center of the exit opening (*f*), one obtains a profile of the drop in pressure head along the pipe; this is the *hydraulic gradient* line. The *energy gradient* line, which represents the rate of decrease of total head, will be obtained by plotting the friction losses (h_f) at all points from the line *A* downward. Since the line thus obtained will be above the hydraulic gradient by a distance equal to $v^2/2g$, which is constant, it will denote the total energy available at any given point. It will be noted that under conditions of steady flow and uniform conduit dimensions these two lines are straight and parallel.

Regardless of the comparative dimensions of the conduits, if fluid enters one conduit from another through a *sharp-edged orifice*, a converging of the stream lines takes place and turbulence is set up (Figs. 12 and 13). Both the energy and hydraulic gradients drop sharply and the velocity of flow as well as the

volume flow decrease. This loss (the so-called *entrance loss*) is distributed along several diameters of the conduit. If the orifice is made bell-mouthed, the loss of flow is less. In sudden *contraction* of the conduit, the turbulence that ensues causes a drop in both energy and hydraulic gradient, the latter being additionally lowered by the increased velocity (Fig. 14). This loss can likewise be reduced by making the constriction in the conduit gradual; an angle of 20 to 40 degrees is considered to be an effective taper. In sudden *enlargement* the loss is mainly in the energy gradient due to both turbulence and decreased velocity (Fig. 15). The hydraulic gradient rises less than one would expect from the increase in the diameter of the conduit. The net result is a decrease in volume flow. The effect can be reduced by making the enlargement a gradual one. The most effective taper under these conditions is one of 6 to 8 degrees.

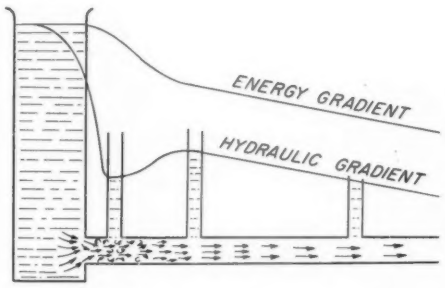


Fig. 12.

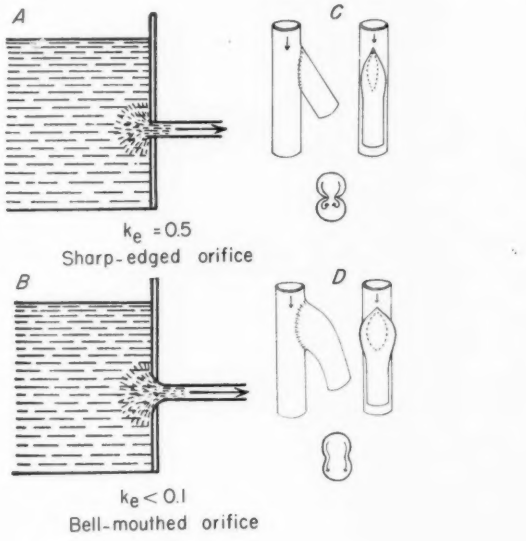


Fig. 13.

Fig. 12.—Entrance loss. Because of the turbulence due to convergence of the stream, when fluid passes through a sharp-edged orifice, a drop occurs in both the hydraulic and energy gradient and the volume flow diminishes. (Redrawn by permission from Daugherty and Ingersoll.[5])

Fig. 13.—The loss of volume flow caused by the conditions of entrance through a sharp-edged orifice (*A*) can be reduced by making the orifice bell-mouthed (*B*). (K_e is the coefficient of entrance loss.) On the right is shown the diagrammatic appearance of an end-to-side anastomosis when the opening in the wall of the host artery is a simple slit (*C*) and when, through the removal of a brim of tissue, it is made bell-mouthed (*D*). (Redrawn by permission from Daugherty and Ingersoll.[5])

A *bend* in a conduit creates turbulence and loss of flow rate because of the disturbance in pressure head brought about by the abrupt change in the direction of the flow (Fig. 16). Owing to the change in flow direction an increase in the pressure head takes place along the outer wall of the bend and a decrease in the pressure head along the inner wall. Since the velocity along the boundary (that is directly at the wall) is zero, it cannot decrease further to provide the required pressure along the outer wall. The unbalanced centrifugal force of the fluid just inside the boundary produces a secondary flow outward along the plane of the bend and inward around the boundary. Thus the fluid moves in double spiral which may persist for as much as 100 conduit diameters downstream. The

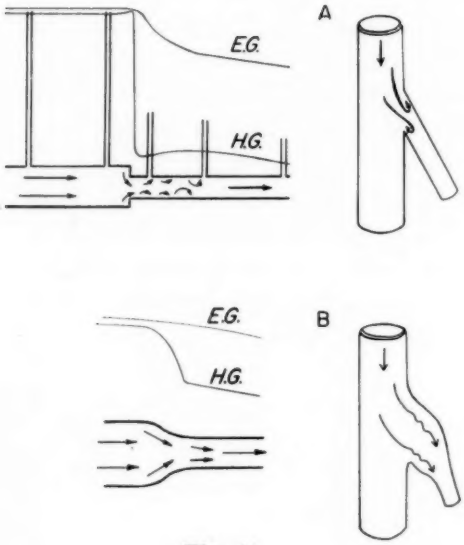


Fig. 14.

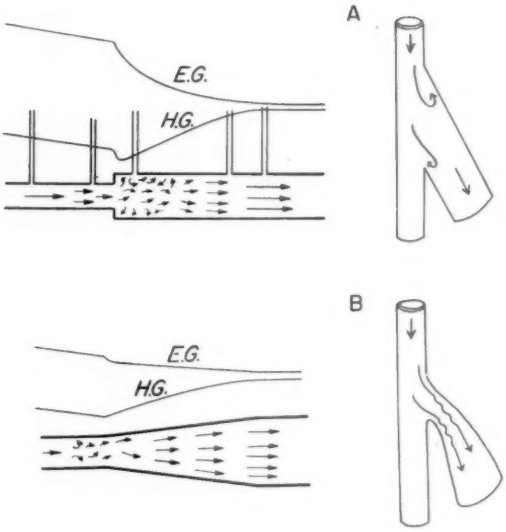
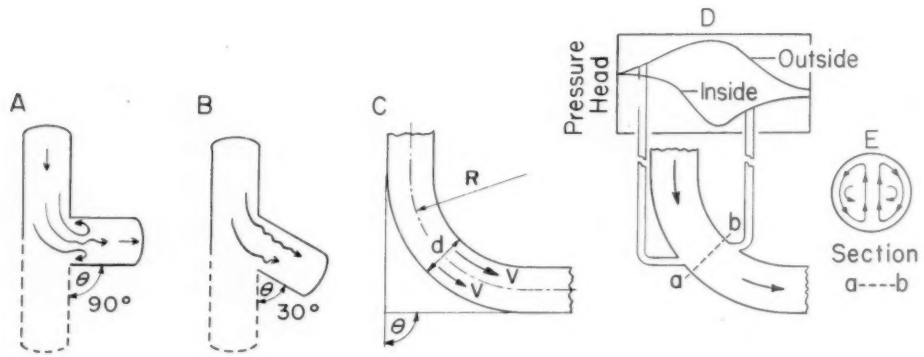


Fig. 15.

Fig. 14.—Contraction loss. At a point of sudden contraction, turbulence leads to loss of flow rate. The energy gradient, instead of rising, drops slightly and the hydraulic gradient falls more than the reduction of pipe diameter would warrant (*A*). This loss can be reduced by making the constriction a gradual one (*B*). The sketches of anastomoses on the right are intended to reproduce the dimensional relationships in the conduit in terms of arterial anatomy and have no reference to actual surgical technique. (Redrawn by permission from Daugherty and Ingersoll.[5])

Fig. 15.—Enlargement loss. At a point of sudden enlargement, the onset of turbulence causes reduced volume flow. The energy gradient drops more and the pressure gradient rises less than predicted from Bernoulli's theorem (*A*). This loss can be lessened by making the enlargement gradual (*B*). The sketches of anastomoses on the right are intended to reproduce the dimensional relationships in the conduit; they have no reference to actual surgical technique. (Redrawn by permission from Daugherty and Ingersoll.[5])



$$\text{Elbow Loss} \propto R/d$$
$$\propto \measuredangle\theta$$

Fig. 16.—Elbow loss. An arterial end-to-side anastomosis, in the hydrodynamic sense is an elbow or bend (*A* and *B*). In a bend, because of the differences in velocity (v in *C*) and pressure head (*D*) along the outer wall, compared to the inner wall, secondary currents are set up (*E*) and the rate of flow is reduced. This loss of flow is proportional to the size of the angle of the bend and to the ratio of the bend radius (R) to pipe diameter (d).

loss of flow rate is proportional to the angle of the bend and to the ratio of the radius of the bend to the pipe diameter. In general, the smaller the angle of bend and the wider the pipe, the smaller is the loss.

Interpretation of the Findings in Terms of Fluid Mechanics.—From the point of view of fluid mechanics the factors that may be assumed to become operative when reconstructive surgical procedure of the type studied in this investigation are carried out are dependent upon the changes in the physical properties of the vascular tube at the site of anastomosis. To list the most important of these changes, such an operation creates new orifices. In an end-to-side anastomosis, a bend or elbow is placed in the path of the blood flow, and, according to the size of the prosthesis chosen, a sudden enlargement or constriction in the effective dimensions of the conduit causes changes in the flow characteristics called entrance, exit, and friction losses, and losses of enlargement and constriction, already briefly described. In most instances there are several factors at work and the resultant change in flow will be the sum of their combined effects. The ultimate flow characteristics will obviously be complex, and this study cannot pretend to furnish their quantitative description. A qualitative analysis of these characteristics, however, seems both valid and instructive.

Little comment is needed on the effect of an end-to-end anastomosis. If the component vessels are of the same dimensions and the line of suturing smooth, only a negligible loss of flow occurs. The only factor reducing flow is the surface roughness created by the suture line which under appropriate technical conditions is very small.

In end-to-side anastomoses, the special parameters to be considered differ according to whether the component vessels are of the same diameter or of different diameters. In an end-to-side anastomosis between an artery and a graft of equal diameter the factors at work are (1) the angle of union of anastomosis and (2) the shape of the new orifice. Since such an anastomosis is a hydraulic elbow or bend, the loss of flow is proportional to the angle of the bend as was pointed out previously; the experimental results were in keeping with this principle. The smaller the angle between the component vessels in the experimental anastomoses, the larger was the flow. An end-to-side anastomosis constructed through a simple slit in the arterial wall represents a sharp-edged orifice with a corresponding serious loss of flow. In the experiments in which bell-mouthed orifices were made by excising a crescent-shaped portion of the arterial wall (thus eliminating the sharp edge of the orifice), this loss could be markedly reduced, a finding that is in complete harmony with the principles of fluid mechanics already stated. These observations undoubtedly hold also for the case in which the vessels making up the end-to-side anastomosis differ in diameter, but these particular cases were not investigated in the study reported here, since in the experiments relating to differences of graft diameter, for the sake of simplification, the anastomoses were all bell-mouthed and similar also in other ways.

In interpreting the observation that the increase of flow rate observed with the increase of graft diameter was not in keeping with the prediction of Poiseuille's law, one must keep several facts in mind. Regardless of whether or

not Poiseuille's equation is directly applicable to the flow of blood in the normal human vascular system (and most likely it is not), in the experimental grafts the increase in diameter of the tube was only one of the important flow factors to be considered. Another and apparently more important parameter in these experiments was the turbulence created by the sudden enlargement of the conduit which tended to reduce the rate of flow. The change of flow rate observed was the resultant of these two opposing variables. As the diameter of the graft was increased beyond the factor of 2, the turbulence became so marked that the volume flow began to diminish. It must be pointed out, further, that the effect of turbulence in these preparations undoubtedly was accentuated owing to the shortness of the experimental grafts. In actual clinical conditions the much greater length of the graft may provide an opportunity for the turbulent flow to be dissipated and for laminar flow to be re-established which, although it does not mean recovery of pressure or volume flow, will affect the flow conditions at the distal anastomosis. For, if an end-to-side technique is used, at the distal anastomosis essentially the same factors come into play that are observed at the proximal anastomosis, creating losses in flow comparable to the loss previously discussed. In short grafts of the end-to-side type, the losses of the near and far sites of the anastomosis may summate, a phenomenon that undoubtedly played a part in the experimental findings. It should be stressed that, while these unavoidable experimental conditions may have exaggerated the losses recorded, they do not in any way alter the fundamental significance of the results.

The behavior observed in smooth and crimped plastic prostheses must be evaluated in terms of surface friction. In general, any irregularity of the surface will result in loss of volume flow, and the magnitude of the loss varies in accordance with the type and relative size of irregularity. The physical properties of the prostheses tested varied considerably in this regard. The greatest losses were noted with prostheses that had a corrugated inner surface or an inner surface with irregularly wrinkled folds. One prosthesis, although crimped, had an essentially smooth inner surface in the relaxed state and showed minimal loss of flow (Fig. 10, *B*). It should be mentioned, however, that the dimensional limitations of the experimental preparations made it necessary to insert this prosthesis loosely; since in clinical use the prosthesis must be implanted under greater tension, which would considerably increase its surface roughness, the experimental results in this instance are subject to some correction. In general, the same remark may be applied, in a lesser or greater degree, to all crimped prostheses.

Clinical Applicability of the Experimental Observations.—It would be most desirable to check these experimental findings, as they are transferred to clinical use, by blood flow measurements at the operating table. Until reliable and simple flowmeters suitable for such investigation are available, however, this important study cannot be performed. Nevertheless, an attempt has been made, in an empirical way, to apply the lessons learned from the experiments. The modifications of technical steps that came from these attempts have been in no way radical but seem to have advantages.

The experimental observations, while pointing out the great efficacy of end-to-end anastomosis, have, for instance, greatly lessened our concern about the hemodynamic performance of end-to-side anastomoses. When no special clinical reasons exist to dictate otherwise, and end-to-end anastomosis with dimensional similarity between host and the graft is undoubtedly the technique of choice. Needless to say, in most clinical instances, however, the pathologic and anatomic conditions demand the use of end-to-side anastomoses. Under these conditions care is exercised to shape the anastomosis in accordance with the concepts discussed. The component vessels are placed at as small an angle as possible, and the orifice in the wall of the host artery is made, whenever possible, a bell-mouthed one by excising an ellipse from the wall. Unfortunately in many cases the diseased state of the arterial wall makes this last-named maneuver impractical. In such instances, one may force the slit to open up to its greatest width by making the bevel of the cut end of the prosthesis slightly longer than the slit in the arterial wall and by taking up the excess cloth by placing the stitches more widely on the prosthesis than on the artery.

In regard to the relative diameters of the graft and the artery, the experimental observations would suggest that the former should be about twice the latter in order to obtain the maximum volume flow. In practice, however, other considerations enter into the decision regarding the optimal graft size. The importance of the flow loss subsequent to enlargement of the conduit has already been discussed. Any increase in the diameter of the graft over the host artery will, in addition, reduce the velocity of flow, which in turn will increase the danger of thrombosis. (In fact, in course of the experiments with grafts of various widths, the incidence of clotting complications was higher the greater the diameter of the graft became.) Moreover, the long arterial segments with which the surgeon deals present a gradual reduction in the size of the artery distalward which—especially in the lower extremity—may be as much as 60 or 70 per cent. A graft of the order of the size twice that of the proximal host artery may be thus excessively wide for the distal arterial segment both from the point of view of technical ease and hemodynamic efficiency. In general, a ratio of 1:1.4 to 1:1.6 between the proximal artery and the graft seems a reasonable compromise. In choosing the low or high ratio, one takes into consideration the degree of tapering from the proximal to the distal arterial segment.

With respect to surface characteristics, it seems clear that a prosthesis with smooth inner surface has a distinct hydrodynamic advantage. This advantage is not of a great magnitude. It is also obvious that in choosing a prosthesis for clinical use other criteria besides textile construction have to be weighed. If, however, from the point of view of the two most important attributes—durability and arteriogenesis—two prostheses are of comparable quality, the one with less surface roughness is preferable.

SUMMARY AND CONCLUSIONS

In the animal laboratory under conditions in a measure approximating the milieu of the operating room, an investigation was made of some of the hemodynamic problems posed by angioplastic procedures. A canine preparation was

used in which one iliac artery served for the experimental flow study while the other iliac artery served as control. The various arterial prosthetic replacements were implanted in the experimental side and the volume flow of blood was determined by simultaneous measurements at the distal ends of both iliac arteries. Under these conditions experimental factors affecting the blood flow were bilaterally identical except for the planned variable introduced by the arterial substitute. With 200 paired flow determinations in about 60 canine preparations the following factors were studied: (1) type of anastomosis (end-to-end compared to various end-to-side anastomoses); (2) bypass grafting as compared to direct replacement; (3) variations in the diameter of the implant; and (4) luminal surface characteristics of the implant.

The observations that were made can be summarized as follows. (1) A properly constructed end-to-end anastomosis is virtually as efficient in transmitting blood as an intact artery. (2) An end-to-side anastomosis is less efficient hemodynamically than an end-to-end anastomosis but its efficiency to a large extent depends upon the size of the angle between the component channels, and, by making the angle sufficiently small, the performance of this type of anastomosis can be made to come very close to that of an end-to-end anastomosis. By removing a small window from the host arterial wall, thus making the anastomosis bell-mouthed, the efficiency of an end-to-side anastomosis can be further increased. (3) A graft placed as a bypass is less efficient hemodynamically than direct replacement of the same size, but the difference can be rendered negligible by using the appropriate surgical technique for the end-to-side anastomoses. (4) An increase in the diameter of the vascular graft by as much as factor of 2 brings about an increase in blood volume flow which, however, is not proportional to the dimensional increase. An increase in diameter over a factor of 2 may result in a decrease in blood volume flow. The optimal ratio between the host and prosthesis appears to be about 1:1.4 to 1:1.6. (5) A prosthesis with rough or corrugated luminal surface is less efficient than a prosthesis whose wall is smooth.

A nonmathematical interpretation of the experimental findings in terms of fluid mechanics is given and their practical applicability is discussed.

We wish to express our thanks to Mr. William Bowman, Senior Research Engineer, Engineering Research Office, Ford Motor Company, for his valuable help in the hydrodynamic interpretation of the experimental findings.

REFERENCES

1. Bayley, F. J.: An Introduction to Fluid Dynamics, New York, 1958, Interscience Publishers, Inc., pp. 105-132.
2. Bruner, H. D.: Bubble Flow Meter, in Potter, V. R., ed.: Methods in Medical Research, Chicago, 1948, The Year Book Publishers, Inc., Vol. 1, pp. 80-89.
3. Catton, W. T.: Physical Methods in Physiology, New York, 1957, Philosophical Library, pp. 73-183.
4. Cox, G. N., and Germano, F. J.: Fluid Mechanics, New York, 1941, D. Van Nostrand Company, Inc., pp. 163-215.
5. Daugherty, R. L., and Ingersoll, A. C.: Fluid Mechanics, New York, 1954, McGraw-Hill Book Company, Inc., pp. 199-232.
6. Dumke, P. R., and Schmidt, C. F.: Quantitative Measurements of Cerebral Blood Flow in the Macacque Monkey, Am. J. Physiol. **138:** 421, 1943.
7. Phelan, J. T., and Herrick, J. F.: Some Rheologic Principles as Applied to Vascular Surgery, Proc. Staff Meet. Mayo Clin. **33:** 108, 1958.

8. Randall, J. E.: Elements of Biophysics, Chicago, 1958, The Year Book Publishers, Inc., pp. 197-223.
9. Rouse, H.: Elementary Mechanics of Fluids, New York, 1946, John Wiley & Sons, Inc., pp. 183-221, 251-263.
10. Wesolowski, S. A., Sauvage, L. R., Pine, R. S., and Fries, C. G.: Dynamics of Blood Flow in Graft Disproportions and in Normal Blood Vessels, S. Forum 6: 227, 1956.

Discussion

DR. SZILAGYI.—As you saw, the differences that we found when we tested smooth against corrugated prostheses were small. It should be remembered, however, that in the investigation of at least two types of prosthesis, the tests were not entirely realistic. In these instances, in order to make usable experimental preparations, we were obliged to insert the prostheses in a loose state which considerably reduces the surface irregularities. In actual clinical practice, these prostheses must be implanted under some tension, a maneuver that accentuates surface roughness. The reduction of flow rate in the experimental preparations, therefore, is probably short of the true reduction in clinical practice. In any case, however, the loss of flow is not very great. I do not wish to leave the impression that an irregular luminal surface is a major deficiency in a prosthesis. However, if I were to choose between two prostheses, one smooth and the other corrugated but otherwise of equal merit, I would select the one with the smooth surface.

AN ASSESSMENT OF THE VALUE OF ARTERIAL GRAFTING

JAMES A. KEY, M.B., W. G. BIGELOW, M.D., AND EARL P. FARBER, M.D.,
TORONTO, ONTARIO

(From the Cardiovascular Service of the Toronto General and Sunnybrook Hospitals and the Department of Surgery, University of Toronto)

THIS is a report on a 3-year follow-up of 97 femoral arterial grafts, 63 aorto-iliac grafts, and 77 cases of aortic resection for abdominal aneurysm.

The surgery of arterial replacement or bypass has gained tremendous momentum in recent years, largely through the pioneer efforts of De Bakey.[1] It is fitting, therefore, to pause and reflect on what we have done—to see if it pleases us and our patients or disappoints us. This, in essence, is the purpose of this review.

CHRONIC ARTERIOSCLEROTIC OCCLUSIVE DISEASE

Our criteria for investigation as to the possibility of arterial grafting in this group of patients have been: (1) the presence of claudication severe enough to interfere with the patients ability to work, (2) claudication severe enough to interfere *materially* with the patient's enjoyment of life, (3) ischemic rest pain, and (4) the presence of incipient or established gangrene.

We have not advocated operation for impotence when it has presented as the only symptom of occlusive arterial disease probably because of our lack of courage or gentle humanity or both!

Apart from routine clinical assessment, our preoperative investigation included aortography and/or femoral arteriography. We have had no worrisome complications from either procedure.

Through the cooperation of Dr. K. W. G. Brown, one of our cardiologists, many of our patients have had pre- and postoperative treadmill tests to determine the degree of claudication under standard conditions before grafting and the subsequent effect on claudication under the same conditions after operation.

Age and Sex.—As one would expect the highest incidence of occlusive disease was in the 55 to 65 years age group. Our youngest patient was 42 years old and the oldest 80.

The sex ratio in the entire series was 4.5 males to 1 female.

Femoral Grafts (97 Cases).—With few exceptions the femoral grafts have all been of the end-to-side bypass type. Forty-four were arterial homografts and 52 were crimped nylon. There was one autogenous vein graft.

Operation has been considered a complete success if it has resulted in the relief of all claudication and the return of one or both pedal pulses. Operation

Read at the Thirteenth Annual Meeting of the Society for Vascular Surgery, Atlantic City, N. J., June 7, 1959.

This review was supported by a Canadian National Health Grant.

has been considered partially successful if the patient's claudication has been significantly improved even though the pedal pulses may not have returned. All operations falling short of these criteria have been considered failures. Of the 97 grafts, 67.5 per cent were a complete success and 17.5 per cent were considered a partial success, an initial success rate of 85 per cent. There was no essential difference noted between homografts and nylon grafts in terms of initial success rate.

Four significant facts emerge from a consideration of Tables I and II: (a) The initial success rate of grafting using either homograft or nylon is high. (b) The early failure rate is higher with nylon grafts than with homografts. (c) The late failure rate is higher with homografts than nylon grafts. (d) Of 10 patients with femoral occlusive disease who were grafted, approximately 8 were initially successful and 5 were satisfactory at the end of 3 years.

TABLE I. FEMORAL GRAFTS—SUBSEQUENT FAILURES

TIME OF FAILURE	PER CENT
Within 6 mo.	18.0
1 year	8.0
2 years	4.8
3 years	1.6
Total	32.4

TABLE II. FEMORAL GRAFTS—SUBSEQUENT FAILURES IN HOMOGRAFTS VERSUS NYLON GRAFTS

TIME OF FAILURE	HOMOGRAFTS	NYLON
Within 6 mo.	9.0%	27.5%
1 year	9.0%	6.8%
2 years	9.0%	0 %
3 years	3.0%	0 %
Total	30.0%	34.3%

Aortoiliac Grafts (63 Cases).—Of the total number of grafts 41 were homografts and 22 were nylon prostheses. With one or two exceptions the proximal anastomosis was of the end-to-end variety. The distal anastomosis was end-to-end in 28 cases and in 35 cases it was of the end-to-side bypass type.

The criteria for success were similar to those for femoral graft patients. Complete success was achieved in 50.9 per cent and partial success in 24.5 per cent, giving a total of 75.4 per cent of the 63 aortoiliac grafts.

From a consideration of Tables III and IV, four significant facts emerge: (a) The initial success rate in aortoiliac grafts using either homografts or nylon is reasonably high. (b) The early failure rate is higher with homografts than with nylon grafts. (c) The late failure rate with homografts and nylon grafts is about the same. (d) Of 10 patients with aortoiliac occlusive disease who were grafted, approximately 7 were initially successful and 5 were satisfactory at the end of 3 years.

Deaths.—There have been 4 deaths in the immediate postoperative period in the entire series—one following a femoral graft and three following aortoiliac grafts. There have been 3 late deaths from presumed coronary infarction.

TABLE III. AORTOILIAC GRAFTS—SUBSEQUENT FAILURES

TIME OF FAILURE	PER CENT
Within 6 mo.	13.1
1 year	0
2 years	5.0
3 years	2.6
Total	20.7

TABLE IV. AORTOILIAC GRAFTS—SUBSEQUENT FAILURES IN HOMOGRAFTS VERSUS NYLON GRAFTS

TIME OF FAILURE	HOMOGRAFTS	NYLON
Within 6 mo.	19.2%	0 %
1 year	0 %	0 %
2 years	3.8%	8.3%
3 years	3.8%	0 %
Total	26.8%	8.3%

Present Thinking.—By stressing our failures rather than our successes we do not wish to convey the impression that arterial grafting is not a very valuable procedure. We think it is and we know of no other certain way of relieving intermittent claudication other than by a successful graft (or thromboendarterectomy).

Many of our failures can probably be explained on the basis of our early thinking which was "graft wherever success seems possible" when it should more properly have been "graft wherever success seems probable." This is further borne out by noting the success rate in relation to the state of the outflow tract as shown in Table V. The method of grading outflow tract occlusion which we adopted will require elaboration in a subsequent review, but our average success rate was much greater when the outflow tract was good than it was when the outflow tract was poor, especially if there was associated gangrene.

TABLE V. CORRELATION OF SUCCESS RATE AND OUTFLOW TRACT
(Based on 89 cases)

OUTFLOW TRACT	FEMORAL	AORTOILIAC
Good	90% success	85% success
Poor	50% success	50% success

In part also, our failures probably reflect technical errors but some of them almost certainly were due to progression of the underlying disease and its cause, about which we are still largely ignorant.

Failure of a graft may occasionally place the patient in a position which is worse than it was prior to grafting. The more distally a graft is inserted, therefore, the higher is the penalty of failure.

Not a few of our patients were about to retire on full pensions when their arterial disease forced them to abandon work temporarily. We have been able to provide the necessary "borrowed time" by successful grafting and therefore return to work until full retirement age, in a gratifying number of cases.

Physicians admit that they have little to offer in terms of drug therapy to the patient with severe obliterative vascular disease. Bearing in mind therefore that we are treating a symptom of a progressive disease of unknown etiology, arterial grafting for the relief of such symptoms should properly be offered patients as long as they understand the implications of their disease and the present limitations of vascular surgery in producing a good long-term result.

The ideal arterial graft substitute has not yet been found.

Considerably better long-term results can probably be achieved by more careful selection of patients and by paying greater attention to the condition of the outflow tract.

ABDOMINAL ANEURYSMS

The surgery of abdominal aneurysms may yet prove to be one of the most rewarding in vascular surgery. Again much credit must go to De Bakey for his contribution to this field of surgical endeavor.

We have resected 77 abdominal aneurysms during the past 3 years. All have been below the renal vessels. There were 46 cases with symptoms and 10 without symptoms. Four postoperative deaths and 2 late deaths occurred in the group with symptoms. No deaths occurred in the group without symptoms. There were 12 cases of retroperitoneal rupture with 8 deaths and 9 cases of intraperitoneal rupture with 7 deaths.

If the results of unruptured aneurysms, either with or without symptoms, are compared with the high mortality associated with operation for a ruptured aneurysm—especially for those patients in whom the aneurysm ruptured intraperitoneally—it would seem justifiable, in view of the natural life history studies carried out at the Toronto General Hospital and other centers, to advise early surgery for this disease. We feel that the distinction between intraperitoneal and retroperitoneal rupture of an aneurysm is important, for the salvage rate from the former is very small in our hands, whereas in the latter group where the bleeding is still confined within the retroperitoneal tissues results have been gratifying.

Of the 77 cases, the aorta was replaced by homografts in 42 and by crimped nylon bifurcation grafts in 35. There were 3 patients with aneurysm developing in the homografts, 2 of whom died following attempts to deal with this complication.

Present Thinking.—A survey of our results has led us to recommend in dealing with aortic aneurysms that: (1) if ruptured, immediate operation gives the only real hope of continued survival of the patient; (2) if producing symptoms, resection of the aneurysm be considered as a relatively urgent necessity; and (3) if symptomless, the aneurysm be removed as an elective procedure, provided that there is no major complication, that the cardiac and renal functions are satisfactory, and that the patient is not too advanced in years. A patient of 85 who has a symptomless abdominal aneurysm is not a subject for operation but for congratulation—on having lived so long!

It is hoped that this survey taken in conjunction with other long-term follow-up studies such as those of De Bakey[1] and Edwards[2] will help in assessing the value of arterial grafting and the proper place it should occupy in that field of cardiovascular surgery directed toward the treatment of the complications of arteriosclerosis of major arteries.

SUMMARY

1. A 3-year follow-up of 160 femoral and aortoiliac grafts and 77 cases of aortic resection for abdominal aneurysm is reported.

2. Our present thoughts on the value of these procedures in relation to preoperative symptoms, initial success rate, and late failures are presented.

We would like to thank Dr. D. R. Wilson, who was on the Staff of the Cardiovascular Service of the Toronto General Hospital during this 3-year period, for allowing us to include his cases in this combined review.

REFERENCES

1. De Bakey, M. E., Crawford, E. Stanley, Cooley, D. A., and Morris, George C., Jr.: Surgical Considerations of Occlusive Disease of the Abdominal Aorta and Iliac and Femoral Arteries: Analysis of 803 Cases, Ann. Surg. **148**: 306, 1958.
2. Edwards, W. Sterling, and Lyons, Champ: Problems in Surgery of Occlusive Disease of the Aorta and Iliac Arteries, Ann. Surg. **149**: 675, 1959.

(For discussion, see page 101.)

EVALUATION OF LATE FAILURES AFTER RECONSTRUCTIVE OPERATIONS FOR OCCLUSIVE LESIONS OF THE AORTA AND ILIAC, FEMORAL, AND POPLITEAL ARTERIES

E. Stanley Crawford, M.D., Michael E. De Bakey, M.D., George C. Morris, Jr., M.D., and Edward Garrett, M.D., Houston, Texas

(From the Cora and Webb Mading Department of Surgery, Baylor University College of Medicine, and the Surgical Services of the Jefferson Davis, Methodist, and Veterans Administration Hospitals)

EXTENSIVE laboratory and clinical investigation during the past 10 years has led to recognition of the frequent segmental nature of atherosclerotic occlusive lesions in patients with chronic arterial insufficiency of the lower extremities and development of techniques to attack these lesions directly. Several procedures [1-3, 5-7] utilizing a variety of arterial replacements in the treatment of this disease have evolved since the early work of dos Santos[4] with endarterectomy. The high percentage of good initial results obtained by continued improvement in technique and increasing surgical experience has led to treatment of many patients with occlusive lesions of the aorta and iliac, femoral, and popliteal arteries by reconstructive operations during the past 6 years.[2, 3, 5, 6] Although pulsatile blood flow in the arterial bed distal to the occlusion has been restored and symptoms of arterial insufficiency have been relieved in most patients, long-term postoperative observation has revealed a significant number of recurrent difficulties.[1, 5, 6] Our experience with this problem in the treatment of 1,225 patients with occlusive lesions of the distal aorta, and iliac, femoral, and popliteal arteries during the last 6 years was reviewed in an attempt to evaluate this form of therapy to discover possible causative technical surgical factors, and to evaluate the therapeutic measures employed for recurrent occlusion.

CLINICAL MATERIAL

The lesions were located in the distal aorta and iliac arteries in 638 of the 1,225 patients and in the femoral and popliteal arteries in 587. The indications for operation were rest pain or intermittent claudication (which was frequently disabling) in 80 per cent of the patients and in the remaining 20 per cent cutaneous lesions that posed an immediate threat to life. Patients were selected for operation on the basis of operative risk and demonstration of localized disease. The technique and immediate results of operation employed in these cases varied with the location and extent of the disease and the quality of the arterial replacement available at the time of treatment as indicated in the following discussion.

Aortoiliac Occlusion.—Lesions of the aortoiliac region were treated by thromboendarterectomy, excision and graft replacement, excision and graft replacement combined with bypass, or bypass graft alone (Table I). Almost all of the earlier patients in the

Supported in part by grants from the U.S. Public Health Service (H-3137) and the Houston Heart Association.

Presented at the Thirteenth Annual Meeting of the Society for Vascular Surgery, Atlantic City, N. J., June 7, 1959.

series were treated by excision and graft replacement. Later, selection of the procedure to be employed depended upon the nature and extent of the occlusive process. Thus, a well-localized process associated with minimal medial mural involvement was treated by thromboendarterectomy. Localized lesions with more extensive medial and adventitial involvement were excised and continuity was restored by graft replacement. Localized lesions with extensive distal involvement were treated by excision with graft replacement and bypass. Under these circumstances the iliac segments of the aortic bifurcation graft were brought down and anastomosed end-to-side to a patent segment of the distal arterial bed, such as the external iliac, femoral, or even the popliteal artery. The bypass procedure without resection or endarterectomy was employed for extensive occlusive lesions involving the distal aorta extending well into the external iliac arteries. In most of these cases aortofemoral bypass was performed, but in some, because of associated segmental occlusion in the superficial femoral artery, it was necessary to extend the bypass to the popliteal artery. An important consideration in these latter cases is the fact that the profunda femoris, along with a small segment of the common femoral artery opposite its opening, is usually patent. In order to improve circulation in the tissues supplied by the profunda femoris and to provide additional runoff for the aortic graft, side-to-side anastomosis was performed between the graft and the patent segment of the common femoral artery immediately opposite the opening of the profunda femoris as well as between the graft and the popliteal artery.

TABLE I. RESULTS IN 638 CASES OF AORTOILIAC OCCLUSION ACCORDING TO TYPE OF OPERATION

		IMMEDIATE SUCCESS		LATE FAILURE				
						REOPERATION		
							SUCCESSFUL	
PROCEDURE	CASES	CASES	PER CENT	CASES	PER CENT	CASES	CASES	PER CENT
Excision and graft replacement	148	141	95	19	13.0	19	17	90
Endarterectomy	121	116	96	3	2.4	3	3	100
Excision and graft replacement with bypass	161	153	95	8	5.2	8	7	87
Bypass only	208	201	97	8	3.8	6	5	83
Total	638	611	96	38	6.2	36	32	91

The types of grafts employed in this group of patients are shown in Table II. Freeze-dried aortic homografts were used predominantly in our early experience. The various synthetic tubes, principally the Edwards-Tapp braided nylon tube, were used from about June, 1956, to the early part of 1957, with satisfactory results. However, all had certain disadvantages resulting from their availability or construction and physical characteristics which limited their adaptability to specific problems and increased the difficulties in their technical application. To overcome these obstacles a flexible, knitted, Dacron tube was developed and this has been used almost exclusively since January, 1957.

TABLE II. RESULTS OF TREATMENT OF AORTOILIAC OCCLUSIONS ACCORDING TO TYPE OF GRAFT REPLACEMENT

		IMMEDIATELY SUCCESSFUL		LATE FAILURE				
						REOPERATION		
							SUCCESSFUL	
TYPE	CASES	CASES	PER CENT	CASES	PER CENT	CASES	CASES	PER CENT
Homograft	173	160	92	20	12.0	20	17	85
Other substitute	69	66	96	10	15.0	8	8	100
Dacron	275	269	98	5	1.8	5	5	100
Total	517	495	96	35	6.8	33	30	91

The results of operative correction of aortoiliac occlusion were classified as successful only if pulses were restored. Failures included patients who died and those in whom distal arterial pulsations could not be demonstrated or who subsequently required amputation even though some improvement may have occurred after operation. On the basis of these criteria reconstructive operation was successful in 96 per cent of the cases (Tables I and II). The results of operation varied somewhat with the type of graft employed, ranging from 92 per cent immediate success with homografts to 98 per cent with the newer Dacron tubes (Table II). There were 16 operative deaths (2.5 per cent) in the entire series. Coronary thrombosis caused death in 9 patients, renal failure and uremia in 4, secondary hemorrhage in 2, and pulmonary embolism in one. In the remaining patients early failure was due to extensive occlusive disease in the peripheral arterial bed precluding direct surgical attack in this region. Amputation of an extremity became necessary in 7 of these, all of whom had severe ischemic changes associated with gangrene before operation. Particularly noteworthy is the fact that circulation in the opposite extremity was improved in all of these patients.

Femoropopliteal Occlusion.—In an attempt to restore immediate distal pulsatile circulation in patients with occlusive lesions of the femoral and popliteal arteries, thromboendarterectomy, excision and graft replacement, or end-to-side bypass graft was employed (Table III). During our early experience these lesions were usually treated by endarterectomy or excision and graft replacement. These procedures were soon almost completely abandoned in favor of bypass grafting since they were associated with relatively high morbidity and failure rates, presumably because of the extensive nature of the procedures, the destruction of collateral channels, and the frequent incomplete removal of the occlusive disease. More recently, endarterectomy has been performed only if the disease is well localized to a short segment of artery. Excision is no longer done. The occlusive process usually involved long segments of the femoropopliteal artery. End-to-side bypass graft was thus considered the procedure of choice in 91 per cent of the cases. In most cases the bypass graft extended proximally from the common femoral artery which was rarely involved by the occlusive process to the patent popliteal artery distal to the occlusion. The operation was performed through two small separate incisions which were connected by a tunnel made by blunt dissection. Among the substances used for vascular replacement were reconstituted freeze-dried arterial homografts, tubes made of compressed Ivalon sponge, braided nylon tubes (Edwards-Tapp), and flexible knitted Dacron tubes (Table IV). These substances were employed during the same periods for reasons previously described in the treatment of aortoiliac occlusion. The Dacron tube was the most satisfactory and was used in 356 patients or 61 per cent of the entire series.

TABLE III. RESULTS OF TREATMENT OF FEMOROPOPLITEAL OCCLUSION ACCORDING TO TYPE OF OPERATION

		IMMEDIATE SUCCESS		LATE FAILURE				
						REOPERATION		
							SUCCESSFUL	
PROCEDURE	CASES	CASES	PER CENT	CASES	PER CENT	CASES	CASES	PER CENT
Endarterectomy	29	20	69	3	15	1	1	100
Excision and graft replacement	19	16	84	14	87	3	3	100
Bypass	539	479	89	92	20	60	54	90
Total	587	515	88	109	21	64	58	91

Distal pulsatile circulation was maintained until after discharge from the hospital in 515 (88 per cent) of the 587 patients with lesions at this level (Table III). The incidence and successful results varied somewhat with the type of procedure and vascular replacement employed. For example, only 20 of the 29 patients (69 per cent) treated by

TABLE IV. RESULTS OF TREATMENT OF FEMOROPOPLITEAL OCCLUSION ACCORDING TO TYPE OF VASCULAR REPLACEMENT

REPLACEMENT	CASES	IMMEDIATE SUCCESS		FOLLOW-UP MONTHS	LATE FAILURE		REOPERATION		
								SUCCESSFUL	
		CASES	PER CENT		CASES	PER CENT	CASES	CASES	PER CENT
Homograft	83	71	85	24-66	37	52	13	12	92
Braided nylon	113	98	86	20-37	32	32	20	19	95
Ivalon	6	0	0	0	0	0	0	0	0
Knitted Dacron	356	326	91	0-25	37	11	30	26	86
Total	558	495	89	0-66	106	21	63	57	91

endarterectomy, and 16 of the 19 patients (84 per cent) who had excision and graft replacement had successful restoration of circulation. Circulation was restored in 479 of the 539 patients (89 per cent) treated by end-to-side bypass. Operation was unsuccessful in all 6 patients in whom an Ivalon tube was used in the bypass graft. Circulation was restored in 85 per cent of patients in whom homografts were employed and 86 per cent of those in whom the Edwards-Tapp tube was inserted. The best results were obtained with the Dacron bypass graft; restoration of circulation was successful in 326 of the 356 patients (91 per cent) in whom this material was employed. Major amputation was required in 11 of the failures (2 per cent) and 3 patients died.

Failure was due to the extensive nature of the disease in most instances; however, the extent to which this led to early failure was related to the type of procedure employed. For example, failure occurred in operable cases (by current standards) in the earlier cases of endarterectomy and excision and graft replacement. During this period, operation frequently consisted of resection or endarterectomy limited to the region of major occlusion without regard for obstructing lesions proximal and distal to the operative site. These partial occlusions were frequently significant, causing retardation of blood flow in the region of operation, early thrombosis, and failure (Fig. 1). In more recent cases in which operation failed, the disease was diffuse, being associated with partially obstructed distal segments and secondary occlusion in the lower leg beyond the present scope of operation.

METHOD OF STUDY

Most of the patients in this series have been followed since discharge from the hospital by personal examination and the others by correspondence with the patient, his family, or his physician. Difficulty relating to the circulation of the lower extremities recurred from one week to 66 months after discharge from the hospital in 38 of the 611 patients (6.2 per cent) with aortoiliac occlusion and 109 of the 515 (21 per cent) with femoropopliteal occlusion in whom operation had been initially successful. Of these 147 late failures, 121 were studied, 38 with aortoiliac and 83 with femoropopliteal recurrent difficulty. Angiograms made before the original operation and patients' charts, including diagrammatic sketches of the operative technique employed, were available in all cases. Angiograms made after failure were available in 105 cases and similar studies performed serially before operation and before and after late failure were available for analysis in 35 cases. Finally, the operative findings in 100 cases submitted to reoperation for late failure were also analyzed and correlated with the other studies.

RESULTS OF STUDY

The incidence, etiology, and rate of late failures varied with the location of the original lesion, the period during which operation was performed, the procedure employed, and the type of material used for graft replacement. The incidence of recurrent difficulty was more than three times greater in patients

with femoropopliteal lesions than in those with occlusion of the aortoiliac segment. This extreme difference justifies division of the problem again into these two groups for purposes of discussion.

Aortoiliac Lesions.—The late failures in the 38 patients in whom reconstructive operations had been successful initially for atherosclerotic occlusive disease of the aortoiliac segment were due to a variety of late complications (Tables V and VI). Failure was due to development of false aneurysm at the site of distal anastomosis in 3 (7 per cent), erosion between the graft site and duodenum in 6 (16 per cent), aneurysmal deterioration of the graft in 2 (5 per cent), and recurrent obstruction in 27 cases (71 per cent) (Figs. 2 and 3). The latter complication was due to kinking of the graft in 3 (7 per cent), presence of distal disease preoperatively in 10 (26 per cent), and progressive narrowing and

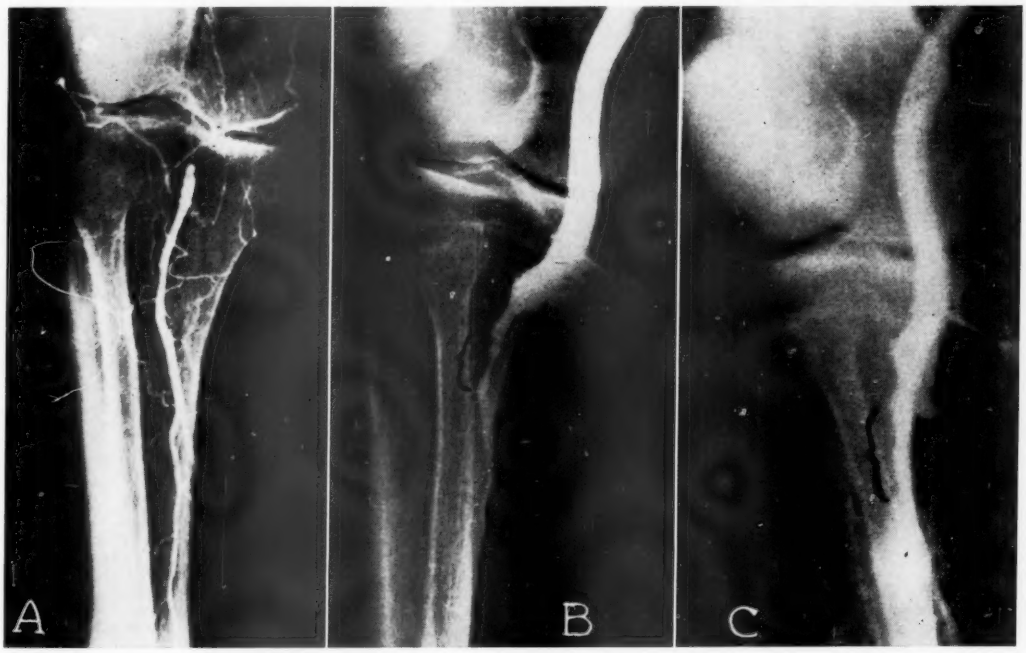


Fig. 1.—Photographs illustrating immediate operative failure because of distal occlusive disease. *A*, Preoperative arteriogram showing distal occlusive disease (*in brackets*). *B*, Arteriogram made immediately after removal of thrombus from graft again showing distal occlusion which caused obstruction and thrombosis. *C*, Arteriogram made after endarterectomy and patch graft closure of arterial incision permitting good outflow and eventual success of operation.

eventual thrombosis in 5 (13 per cent) in whom anastomotic narrowing had been present from the time of the original operation (Fig. 3). The cause of recurrent obstruction in the remaining 9 patients (24 per cent) was not entirely clear; however, correlation of the observations at reoperation with the aortograms made before the original operation and after failure suggested that obstruction was due to distal disease in the external iliac arteries overlooked at the time of operation. The interval between operation and late failure ranged from 1 to 52 months (Table V). Although this interval did not vary significantly with the complication causing failure, the longest period of temporary function was in the patients in whom recurrent obstruction developed from progression of untreated

disease. The first 2 years after operation were the most critical from the standpoint of recurrent difficulty, 69 per cent of the complications developed during this time. The incidence of recurrences after 2 years was indeed small.

The incidence of failure varied with the type of procedure performed and the material employed as the vascular replacement. For example, late failure

Fig. 2.—Aortogram and femoral arteriograms made 21 months after aortic excision and replacement with bypass graft of braided nylon, showing false aneurysms (*arrows*) at suture line between graft and common femoral arteries.

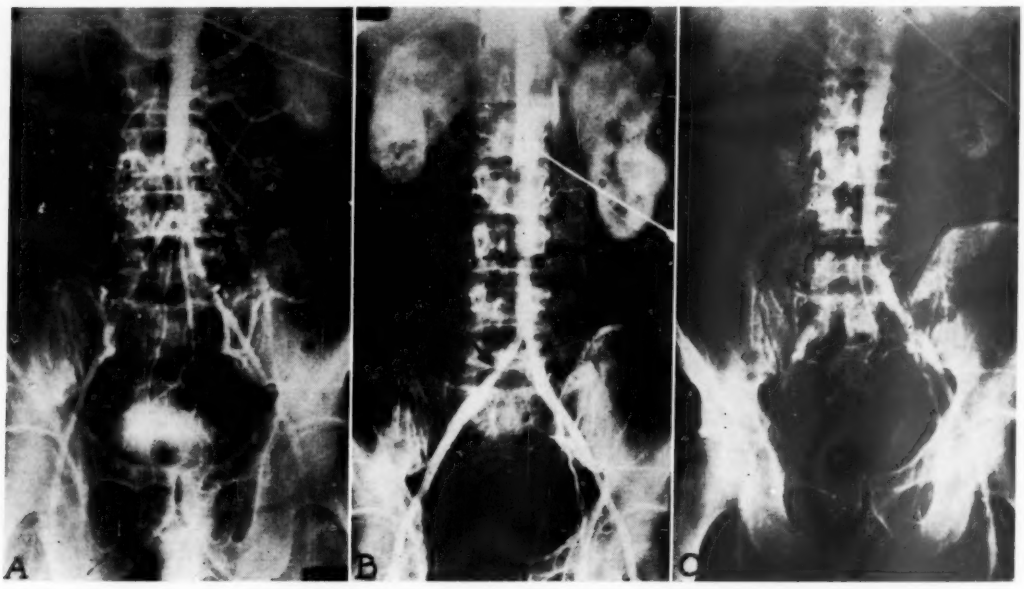


Fig. 3.—Photographs illustrating progressive narrowing at anastomotic site after excision and replacement with homograft. *A*, Preoperative aortogram showing complete occlusion of distal aorta. *B*, Aortogram 30 months after operation showing stenosis (*arrows*) in region of anastomosis. *C*, Aortogram 54 months after operation showing progression of obstruction, complete on right and incomplete on left.

TABLE V. CAUSES OF LATE FAILURE AND REOPERATION IN 38 PATIENTS WITH AORTOILIAC OCCLUSIONS

CAUSES OF FAILURE	CASES	PER CENT	MONTHS POST-OPERATIVE	LESION REMAINED SEGMENTAL	REOPERATION		
					CASES	SUCCESSFUL CASES	SUCCESSFUL PER CENT
Aortoduodenal fistula	6	16	2-30	6	5	4	80
Aneurysm of graft	2	5	18-24	2	2	1	50
False aneurysm	3	7	1-22	3	3	3	100
Kinked graft	3	7	5-23	3	3	3	100
Distal disease	10	27	3-38	10	10	10	100
Narrowed anastomosis	5	13	3-52	5	5	5	100
Recurrent obstruction (cause unknown)	9	24	2-46	9	8	7	88
Total	38	100	1-56	38	36	33	91

after excision and graft replacement occurred over three times more frequently than after endarterectomy or operations employing the bypass principle (Table I). Late failure was rare in patients in whom the flexible, knitted, Dacron tube was used: only 1.8 per cent of the cases (Table II) as contrasted with 12 per cent in patients with homografts and 15 per cent in those who had synthetic materials other than the Dacron tube. These differences in failure rate were also related significantly to the time in the series when the operation was performed as well as to the physical characteristics of the vascular replacement. Both excision and replacement with a homograft were employed almost routinely in the earlier cases, frequently without regard to partial involvement of the more distal iliac arteries. During this period a precise method of end-to-end anastomosis was being evolved, and the need to surround the graft by viable tissues to incorporate the graft, and particularly to separate it from the duodenum, had not been recognized. This combination of factors led to a higher incidence of failure resulting from aortoduodenal fistula, narrowed anastomosis, and recurrent obstruction (Table VI). Aneurysmal deterioration occurred in the femoral segments of two aortoiliofemoral homografts employed early as aortofemoral bypass grafts, again reflecting the limitations inherent in the use of homografts (Fig. 4).

Techniques of operation had been extended and improved to incorporate the bypass principle by the time braided nylon tubes were adopted as the vascular replacement. With increasing surgical experience and better concepts of the

TABLE VI. CAUSES OF LATE FAILURE ACCORDING TO REPLACEMENT IN 38 CASES OF AORTOILIAC OCCLUSION

REPLACEMENT	AORTO-DUODENAL FISTULA	ANEU-RYSM OF GRAFT	FALSE ANEU-RYSM	RECURRENT OBSTRUCTION				TOTAL
				NAR-ROWED ANASTO-MOSIS	KINKED GRAFT	DISTAL DISEASE	CAUSE NOT PROVED	
Homograft	4	2	0	5	0	5	4	20
Dacron	1	0	0	0	1	2	1	5
Braided nylon (Edwards-Tapp)	1	0	3	0	2	0	4	10
No replacement (endarterectomy)	0	0	0	0	0	3	0	3
Total	6	2	3	5	3	10	9	38

pathologic process, the late failure rate from distal disease was significantly reduced. However, certain physical characteristics of the vascular replacement resulting from its method of construction introduced other factors that led to a relatively high incidence of late failure. Flexibility was sacrificed and fraying of the cut ends of the graft prevented precise anastomosis. These limitations, inherent in this type of replacement, resulted in development of false aneurysm and obstruction from kinking of the graft (Table VI). Introduction of the crimped knitted Dacron tube increased flexibility and eliminated fraying and its associated production of false aneurysm. Application of the Dacron tube in most of the recent cases as a bypass graft extending from the aorta to the common femoral arteries decreased the possibilities of overlooking distal iliac disease which would later lead to recurrent obstruction. Consideration of these factors resulted in a higher incidence of long-term function with use of the Dacron tube than with the other replacements.

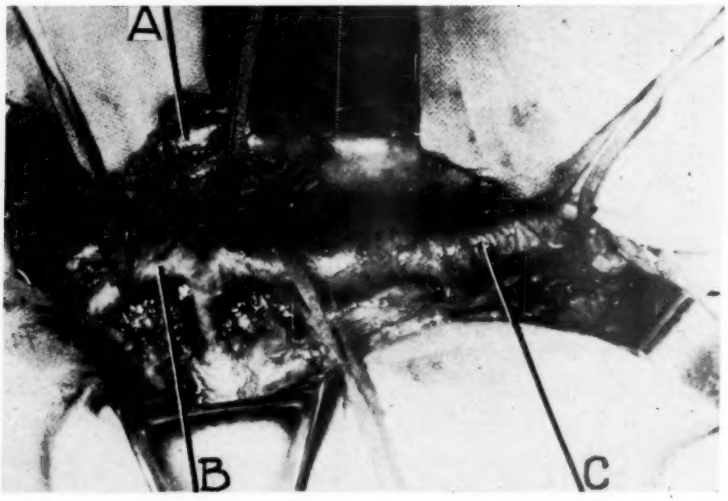


Fig. 4.—Photograph at second operation demonstrating aneurysm of femoral segment of aortoiliofemoral homograft (*A*) employed as aortofemoral bypass, occurring 36 months after first operation. *B*, Superficial femoral artery. *C*, Common femoral artery.

The recurrent lesion in all cases in this series was segmental and 36 patients were reoperated upon. The indications for reoperation were aortoduodenal fistula in 5, false aneurysm in 3, aneurysm of the graft in 2, and recurrent obstruction in 26 cases. One patient with recurrent obstruction declined operation and one patient with aortoduodenal fistula died before operation could be performed. The operative procedure employed in these cases varied with the lesion. The false aneurysms and the aneurysms involving the femoral segments of aortofemoral bypass grafts were located in the inguinal region. These lesions were excised and replaced with braided nylon tubes sutured proximally to the end of the previous graft and distally to the femoral artery at the site of the previous anastomosis. All patients with recurrent obstruction were treated by the bypass principle. The proximal end of the tube was sutured to the side of the aorta above the obstruction and previous site of operation in 25 cases. In the remaining case the partially obstructed homograft was excised and replaced

by a Dacron tube. The distal ends of the tubes in all cases were brought down and attached by end-to-side anastomosis to a patent segment of the distal arterial bed. In 20 cases it was possible to apply the procedure as an aortofemoral bypass. In 6 cases, however, it was necessary to extend the bypass to the popliteal artery because of secondary disease of the superficial femoral artery. In the latter cases side-to-side anastomosis was made between the patent common femoral artery and graft for reasons previously described.

Treatment of aortoduodenal fistula presented special problems concerning primarily the diagnosis, control of hemorrhage during operation, and management of an infected graft site. The diagnosis was suggested by the history of an aortic graft operation and by the symptoms. The predominant symptom was intermittent upper gastrointestinal hemorrhage. The bleeding episodes were mild to severe, occurring over a period of hours to 4 days. An extremely important characteristic of the bleeding was the fact that these patients did not have symptoms of other lesions commonly associated with gastrointestinal hemorrhage, namely, peptic ulcer, esophageal varices, and hiatal hernia. Immediate exsanguination fortunately did not occur in 5 patients, so that the operation necessary for both diagnosis and treatment could be performed. Because of the intimate adherence of the duodenum to the graft site, the diagnosis was not obvious from simple exploration of the abdominal cavity. The fistula and site of bleeding could be demonstrated only by separating the duodenum from the aortic graft. To control hemorrhage during operation circulation through the abdominal aorta was temporarily arrested by placing a clamp across the upper abdominal aorta in the hiatus of the diaphragm. A segment of aorta was then exposed between the renal arteries and the graft site. A clamp was placed across the aorta at this level and the other clamp was removed. With circulation maintained to the abdominal viscera and arrested in the region of operation, the previously inserted grafts were removed. In 3 patients aortoduodenal fistula occurred 12 to 26 months after insertion of a homograft to replace the distal abdominal aorta as a result of erosion by false aneurysm. False aneurysm was due to degeneration of the graft in the region of an unabsorbed unorganized collection of fluid between the duodenum and graft. Infection was not present in these cases; consequently, the duodenal defect was closed by simple suture and aortic continuity was restored by insertion of a new graft. Infection and abscess had caused erosion into the duodenum and the suture line in 2 patients in whom synthetic tubes (Dacron in one and braided nylon in the other) had been inserted as aortofemoral bypasses. Aortic continuity was not restored in these cases because of fear of recurrent infection and secondary hemorrhage. The aorta was ligated by suture above and below the region of anastomosis and the femoral arterial defects were closed by simple suture. Recurrence of fistula was prevented by suturing the omentum between the duodenum and aorta.

The results of reoperation were extremely satisfactory (Tables I, II, and V). Normal peripheral circulation was restored in 33 of the 36 cases (91 per cent). A distal pulsatile circulation was not restored in the 2 patients in whom aortic continuity was not restored after removal of infected synthetic tubes. One of the iliac arteries was incompletely obstructed before operation in one of these

patients and collateral circulation was insufficiently developed to prevent amputation on this side. Circulation in the other three extremities of these patients was adequate at rest. Death from massive retroperitoneal anaerobic infection occurred in one patient who was reoperated on for aneurysm of the femoral segment of an aortofemoral homograft bypass.

Femoropopliteal Occlusions.—Failure occurred after successful reconstructive operation in 109 of the 515 patients (21 per cent) with femoropopliteal occlusions. Although the period of function in these cases ranged from 3 weeks to 56 months, most failures occurred within the first year (Fig. 5). The incidence of recurrent difficulty varied significantly with the extent of the lesion, the type of procedure, and the material employed as a vascular replacement in the operation (Tables III and IV). The best long-term results were obtained with endarterectomy, limited to short, discrete, well-localized lesions; failure occurred in only 3 cases (15 per cent). Despite the fact that the bypass graft was employed in the most advanced and extensive lesions, late difficulty occurred in only 92 of the 479 cases (20 per cent). The poorest results were obtained in patients who had excision and arterial replacement; failure occurred in all except 2 of these. The influence of the graft material on the long-term results is demonstrated by the fact that failure occurred in 52 per cent of the patients with homografts, 32 per cent with the braided nylon Edwards-Tapp tube, and 11 per cent with the flexible, knitted, Dacron tube. Although there has been greater opportunity to observe the long-term functional results of homografts and nylon tubes than Dacron tubes, the length of follow-up does not appear to be the most important factor leading to differences in late failure. For example, analysis of a comparable series of patients followed from 18 to 30 months reveals a significant difference in the percentage of successful long-term results (Fig. 6).

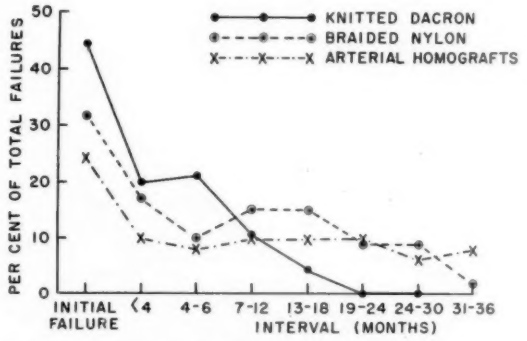


Fig. 5.—Rate of graft failure after operation for femoropopliteal occlusion, comparing functional results of Dacron tubes, nylon tubes, and homografts.

At the end of 18 to 24 months 75 per cent of the Dacron tubes were still functioning in contrast to 64 per cent of the nylon tubes and 60 per cent of the homografts. Similarly, study of the differences in the rate of failure indicates that all failures with Dacron tubes occurred during the first 18 months and that good function was obtained in all cases during the next 12 months (Fig. 5). On the contrary, a small but significant percentage of the nylon grafts and homografts continued to fail in subsequent months, the rate being greater with homografts, despite the fact that most late failures occurred during the

first 24 months. The nature and incidence of the complications leading to failure, as will be described later, were also related to the type of operation performed and the material employed as the graft.

Of the 109 patients with recurrent difficulty after successful reconstructive operation for femoropopliteal occlusions, 20 did not return for examination. Failure of operation was confirmed by examination in 89 patients with arteriography in 83 (76 per cent) which included 2 patients who had endarterectomy, 4 who had excision and graft replacement with homograft, and 77 who had the bypass graft procedure (homograft in 17, braided nylon tube in 26, and Dacron tube in 34 cases). Arteriograms made before operation and after failure were available in 57 cases, and serial arteriograms made before operation and before and after failure were available in the remaining 26 cases. These studies were analyzed and correlated with other available information such as that obtained from reoperation. Reoperation, however, although informative in some cases, did not reveal as much information as desired regarding recurrent occlusions. For example, operation in patients with recurrent obstruction frequently consisted of bypass graft extending both proximal and distal to the previous operation without exposing the latter. This approach often did not permit operative confirmation of the cause of recurrent obstruction.

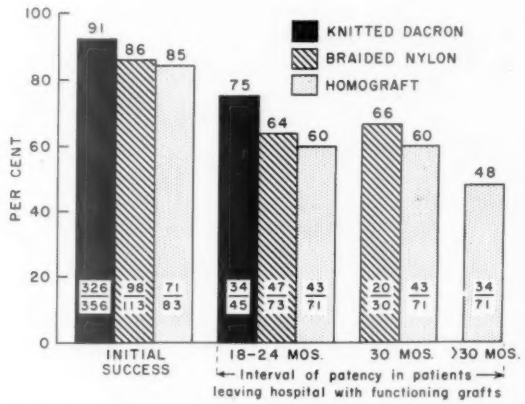
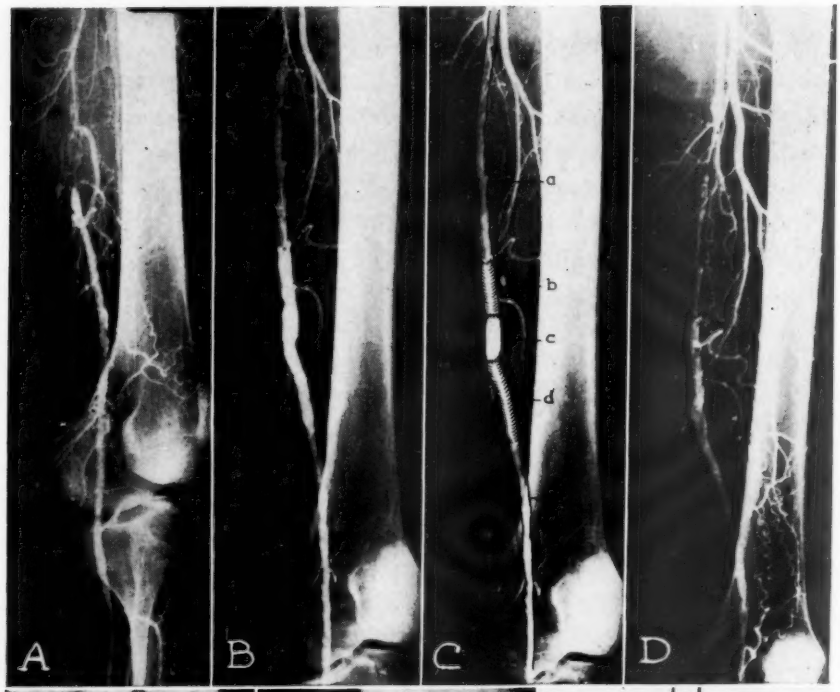


Fig. 6.—Immediate and long-term results according to vascular replacement after operation for femoropopliteal occlusion.

The complication leading to failure was reasonably obvious in most instances (Table VII). Recurrent difficulty was due to certain technical surgical factors in 47 patients (57 per cent), and the underlying disease in 36 (43 per cent). Occlusive lesions causing recurrent obstruction were present in the vessels proximal to the region of operation in 11 patients and distal to the previous operative site in 25 patients. This complication was responsible for failure in all 6 of the patients in whom excision and homograft replacement or endarterectomy had been performed (Fig. 7). These procedures were employed early in our experience when the significance of minimal-to-moderate proximal and distal disease was not appreciated. In the remaining 30 patients in whom this complication occurred after the bypass procedure, the proximal and distal disease was overlooked because of poor quality of the arteriograms leading to improper performance of anastomosis in relationship to the disease. For example,

Fig. 7.



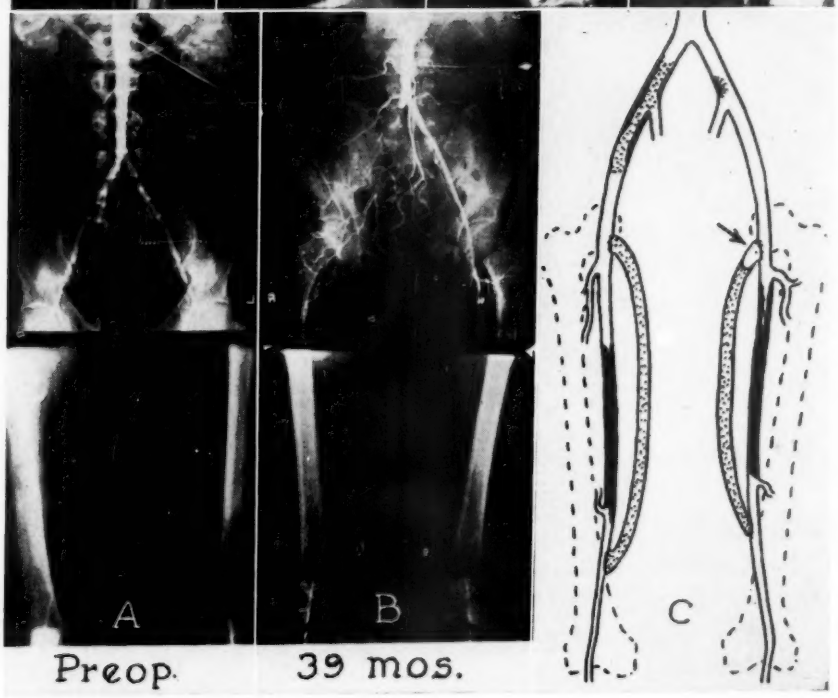


Fig. 8.

Fig. 7.—Arteriograms made before operation (*A*) showing complete obstruction of superficial femoral artery, and immediately after (*B* and *C*), showing excision and homograft replacement (*c*) with endarterectomy (*b* and *d*) leaving partial obstruction (*a* and *e*), becoming complete (*D*) above the region of operation 54 months later.

Fig. 8.—*A*, poor quality aortogram and femoral arteriogram vaguely showing partial occlusion of both iliac arteries overlooked before performing bilateral femoropopliteal bypass. Aortogram made 39 months after operation (*B*) and diagram (*C*) showing proximal complete occlusion of right iliac artery, partial occlusion of left iliac artery, and distal occlusion of both grafts except for small segment at anastomosis on left (*arrow*). Popliteal arteries remain unchanged.

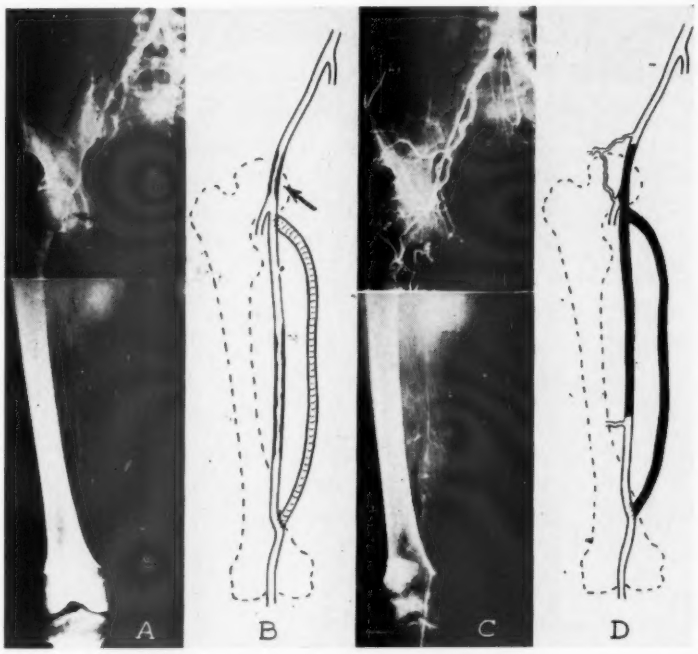


Fig. 9.—Overlooked proximal partial occlusion of right common femoral artery causing late occlusion of femoral bypass graft. *A*, Poor quality preoperative aortogram and femoral arteriogram vaguely showing (*arrow*) partial occlusion of proximal segment of common femoral artery and partial occlusion of right superficial femoral artery. *B*, Diagram of operation showing bypass graft employed distal to occlusion (*arrow*). *C*, Aortogram and femoral arteriogram 4 months after operation showing complete proximal occlusion of common femoral artery and distal bypass graft. Distal popliteal artery remains unchanged. *D*, Diagram of failure showing occlusions in black.

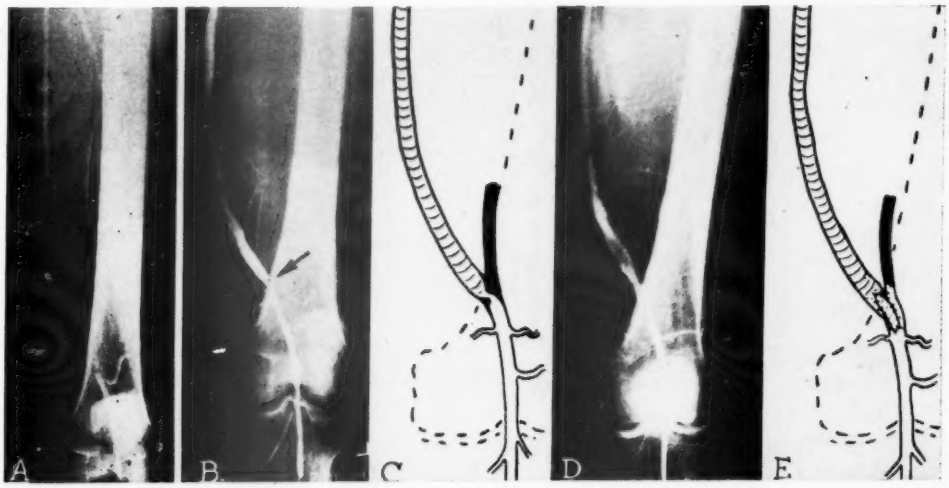


Fig. 10.—Operative failure resulting from performance of distal anastomosis in region of disease proximal to normal patent distal segment. *A*, Preoperative arteriogram showing complete occlusion of distal segment of superficial femoral artery with partial occlusion above prominent collateral vessel. *B*, Arteriogram made 10 months after operation because of persistent symptoms showing partial occlusion at anastomotic site from persistent occlusive disease not bypassed at operation. *C*, Diagram showing outflow from graft partially obstructed by persistent disease (*in black*). *D*, Arteriogram 10 days after removal of obstruction by endarterectomy showing patent channels. *E*, Diagram of operation showing patch graft closure of longitudinal incision employed for endarterectomy.

femoral grafts were inserted distal to the iliac occlusion in some cases, and in others the distal anastomosis was performed above occlusions located in the popliteal artery (Figs. 8-10).

TABLE VII. CAUSES OF LATE FAILURE ACCORDING TO REPLACEMENT IN 83 CASES OF FEMOROPOPLITEAL OCCLUSION

REPLACEMENT	ANEURYSM OF GRAFT	FALSE ANEURYSM	NARROWED ANASTOMOSIS	DISTAL DISEASE	PROXIMAL DISEASE	CAUSES RELATED TO GRAFT	TOTAL
Dacron	0	2	2	11	6	13	34
Braided nylon (Edwards-Tapp)	0	6	4	6	2	8	26
Homograft	4	0	0	7	2	8	21
Endarterectomy	0	0	0	1	1	0	2
Total	4	8	6	25	11	29	83

Fig. 11.—Arteriograms made 1 month after operation showing arterial homograft bypass with minimal narrowing in the proximal anastomosis (*A*), and 56 months after operation showing degeneration of homograft and progression of obstruction at proximal anastomosis (*B*).

The remaining complications resulted from causes related to the graft in most instances and varied with the type of replacement employed in the performance of the bypass procedure. Graft failure occurred in 12 homografts, fusiform aneurysms occurred in 4, and other complications related to the graft, such as dilatation, stricture, and deterioration occurred in 8 cases (Fig. 11). The cause of graft failure in 18 patients who had braided nylon tubes was false aneurysm in 6, narrowed anastomosis in 4, and technical difficulties, such as twisting and kinking, in 8. Graft failure from this cause occurred in 17 patients

who had Dacron tubes, false aneurysm in 2, and mechanisms related to insertion of the tube in 15 cases (Figs. 12-15).

Most failures due to causes relating to the graft, such as kinking, twisting, deterioration, and mechanical difficulties concerned with insertion, were observed at reoperation or were visualized by serial arteriograms. In some, however, this cause was determined from studies that did not include actual visualization of the graft itself. For example, in some patients whose arteriograms of the functioning graft were not available, this was considered the cause of failure because, as in the proved cases, the arterial channels after failure appeared unchanged arteriographically from those preceding operation. This seemed even more reasonable since short segments of the occluded graft could be visualized after failure in the region of each anastomosis, so that progression of disease and anastomotic difficulties could be excluded as causes of failure (Fig. 15). In some patients without serial arteriograms, the distal, previously patent arterial segment was partially or completely obliterated after failure. Reoperation, consisting of transection of the Dacron bypass near its origin and insertion, removal of fresh thrombus from the patient's vessels through the proximal and distal graft segments, and insertion of a second bypass graft sutured proximally and distally to the short segments of the old graft, restored circulation and an arteriographic pattern similar to that before the original operation in 9 cases (Fig. 14). Failure in these cases was also considered to be due to abnormality of the graft. The other cases with similar arteriographic observations not explored in this manner were recorded as failures due to extension of distal disease, although in reality the graft may have been at fault.

Treatment in the 109 patients with recurrent difficulty after femoropopliteal reconstructive operation depended upon a number of factors including whether or not the patient returned for treatment, the extent of symptoms particularly when different from the preoperative ones, and the extent of the occlusive process. The indication for the original operation in these cases was intermittent claudication or mild-to-moderate rest pain in 92 (84.5 per cent), and severe rest pain associated with cutaneous lesions threatening amputation in 17 patients (15.5 per cent). Although these symptoms were relieved in all patients by the initial operation, somewhat different symptoms developed in every patient after recurrent obstruction. For example, recurrent cutaneous lesions developed in only 7 of the 17 patients (41 per cent) who had had this symptom before operation and in only one of the 92 with intermittent claudication. Claudication and rest pain were worse in 8 of the 92 patients (8 per cent) with this symptom before operation. Peripheral circulation and the symptoms of arterial insufficiency were better after an initial period of graft function than before operation in some cases as indicated by this finding in 12 of the 89 patients (13 per cent) who returned for examination and treatment.

Amputation was performed elsewhere in 3 of the 20 patients who did not return for examination, and in the remainder peripheral circulation allegedly remained unchanged. Most patients, with severe recurrent symptoms or whose symptoms became worse, returned as was expected. Of the 89 patients examined, 83 were studied for the possibilities of a second reconstructive operation.

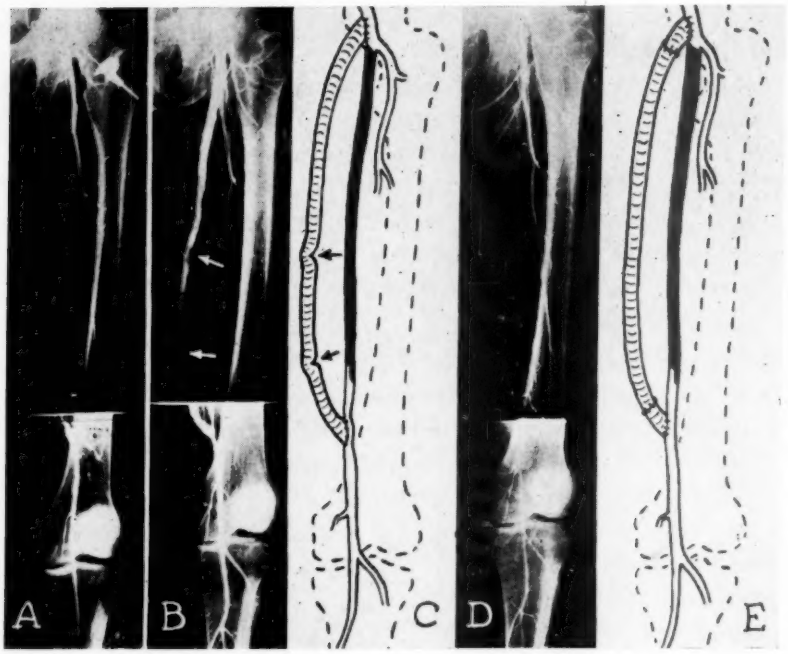


Fig. 12.—Case demonstrating failure from kinking of graft. *A*, Preoperative arteriogram showing extensive complete occlusion of superficial femoral artery. *B*, Arteriogram made 3 months after operation showing kinks (*arrows*) in Dacron graft. *C*, Diagram showing kinks (*arrows*) and occlusion (*in black*). *D*, Arteriogram made 18 months after operation showing occlusion of graft and progression of occlusion into popliteal artery by thrombus. *E*, Diagram showing method of operation in which old graft is transected near previous anastomoses and new graft is inserted after removal of proximal and distal thrombus through short segments of old graft left attached to arteries for new anastomoses.

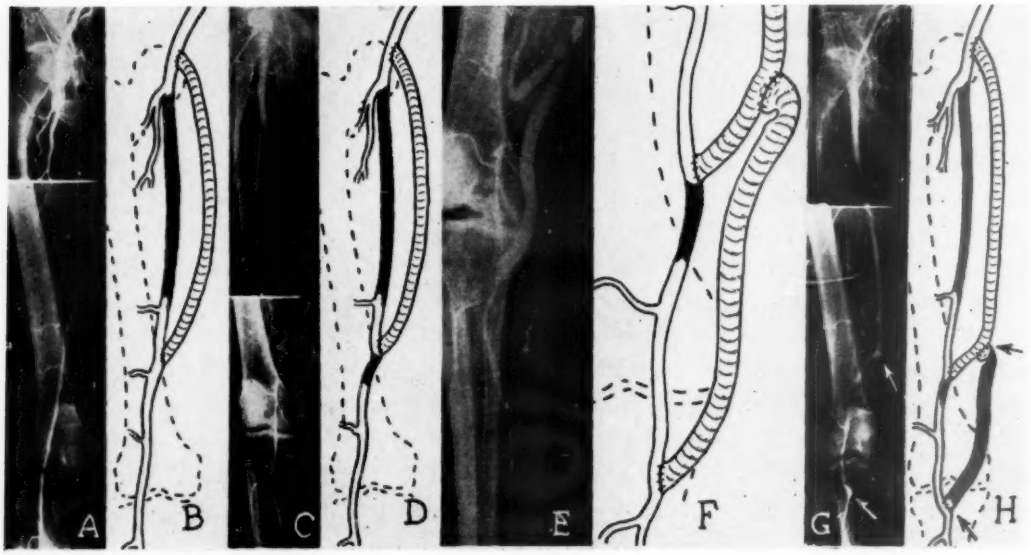


Fig. 13.—Case demonstrating failure from kinking of graft. *A*, Preoperative arteriogram showing complete occlusion of superficial femoral artery. *B*, Diagram of femoropopliteal Dacron graft bypassing occlusion (*in black*). *C*, Arteriogram made 8 months after operation showing occlusion of popliteal artery just distal to anastomosis. *D*, Diagram showing bypass graft and occlusion (*in black*). *E*, Arteriogram made at operation showing kink just distal to proximal anastomosis of new graft to old graft. *F*, Diagram showing bypass graft with kink and obstruction (*in black*). *G*, Arteriogram made 3 months after second operation showing occlusion of second graft and persistent patency of arterial segments. Region of previous anastomosis (*arrows*) is obviously not obstructed. *H*, Diagram showing obstructed graft and site of anastomosis (*arrows*).

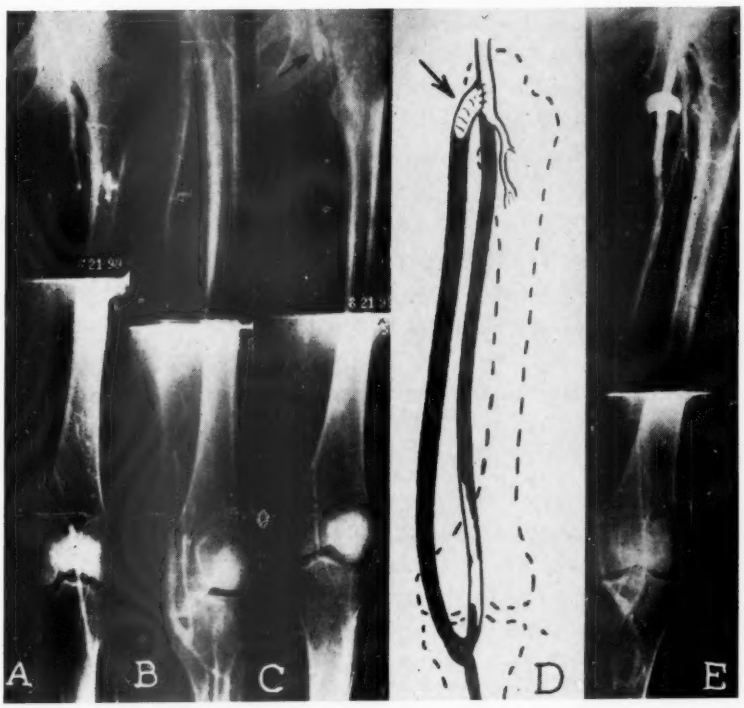


Fig. 14 —Arteriograms and diagram showing occlusion of superficial femoral artery (*A*), functioning Dacron bypass graft 10 days after operation (*B*), occlusion of graft and distal popliteal artery 3 months after operation (*C*), occlusion (*D*) (*in black*), and new functioning bypass graft inserted after thrombectomy through distal end of old tube preserved for site of new anastomosis (*E*).

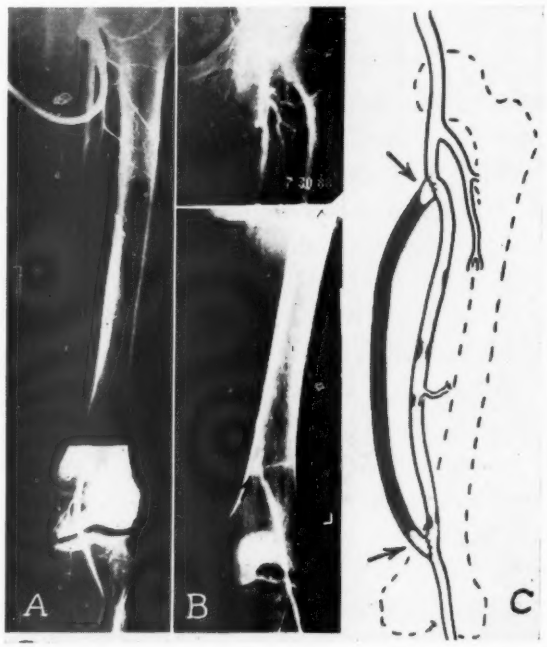


Fig. 15.—*A*, Arteriogram made before operation showing partial occlusion of superficial femoral artery. *B*, Arteriogram made after graft occlusion 17 months after bypass graft operation showing sites of patent anastomoses and unchanged arterial segments indicating occlusion resulting from factors related to tube. *C*, Diagram showing occluded tube and patent anastomoses.

These studies indicated that the lesion remained segmental and amenable to reconstructive operation in 72 cases (86 per cent). In the remaining 11 cases the disease had progressed to involve the major part of the previously patent distal segment. Peripheral circulation at rest was good in 5 of the latter patients; consequently operation was not advised. Sympathectomy was performed in 2 of the patients with diffuse involvement, and, because of the advanced nature of the lesion and the severe associated ischemic changes, amputation was performed in 4 patients. Of the 72 patients with segmental lesions, 8 declined operation because their symptoms were better than before the original procedure; the remaining 65 had a second operation (Table VIII).

TABLE VIII. RESULTS OF REOPERATION IN 64 PATIENTS WITH FEMOROPOPLITEAL RECURRENT DIFFICULTY

COMPLICATION	REGRAFT		
	CASES	SUCCESSFUL	
		CASES	PER CENT
False aneurysm	6	5	83
Anastomotic narrowing	6	6	100
Graft difficulties	28	28	100
Distal disease	12	8	66
Proximal disease	8	7	87
Aneurysm of homograft	4	4	100
Total	64	58	91

The type of secondary reconstructive operation employed depended upon the complication that produced failure of the original procedure. Aneurysms of the homografts and false aneurysms associated with use of synthetic materials were treated by removal of the involved graft and replacement with a new graft. Recurrent obstruction was treated by end-to-side bypass graft extending usually from the proximal aorta, common iliac artery, or common femoral artery to the distal popliteal artery bypassing both the diseased segment and region of previous operation. The popliteal artery was explored in 10 recent patients who were seen soon after graft occlusion despite the fact that arteriograms showed the recurrent occlusive process extending beyond the region of previous operation frequently involving the popliteal artery (Fig. 14). This extension of the occlusive process was found to be due to thrombi. It was possible to remove the thrombus in 9 patients, and peripheral pulsatile blood flow was restored by insertion of a new graft. This indicates that reoperability is not necessarily predictable on the basis of arteriograms and demonstrates the limitations of this method of study in the cause of late failure. Operation consisted first of exposing the graft and popliteal artery in the region of the distal anastomosis. The occluded graft was then transected 2 cm. proximal to the anastomosis and the distal clot was removed from the distal segment of graft. The thrombi removed in this manner were frequently casts of the entire distal popliteal artery. A vigorous backflow of blood was obtained in 9 patients, indicating distal patency. The femoral artery and upper end of the previous graft were then exposed. The graft was transected about 2 cm. distal to its origin and the thrombus was removed from the small segment of graft still attached to the artery. The old

graft was adherent to the tissues in the tunnel and could not be removed; consequently, a new graft was inserted through a new tunnel made by blunt dissection. The two ends of the new graft were sutured to the ends of the short segments of the previous graft which were still attached in the region of anastomosis. Postoperative arteriograms showed good function of the grafts and restoration of patency of the distal segments of the arteries equal to that demonstrated before the first operation (Fig. 14). The distal thrombus could not be removed in one patient because of the organization that had occurred during the 5 days between graft failure and reoperation. This case demonstrated the importance of the time factor in reoperation of patients in whom distal occlusion has become extensive because of progression of the thrombus.

Endarterectomy with patch graft was performed on one patient who had recurrent partial obstruction resulting from progression of disease overlooked at the time of the original operation in the region of the distal anastomosis (Fig. 10). The distal end of the graft and the popliteal artery were exposed through the old incision. A longitudinal incision was made in the distal end of the graft and continued into the popliteal artery for approximately 3 cm. With the occluding lesion thus exposed, endarterectomy was carefully performed under direct vision by entering the proper cleavage plane and removing the diseased intima. The opening in the graft and popliteal artery was closed with a patch graft to prevent narrowing and recurrent obstruction. The edges of the graft and arterial incision were sutured circumferentially to the edges of an oval-shaped patch made from knitted Dacron fabric. The circumference of the vascular channel in this region was increased by a distance equal to the width of the patch.

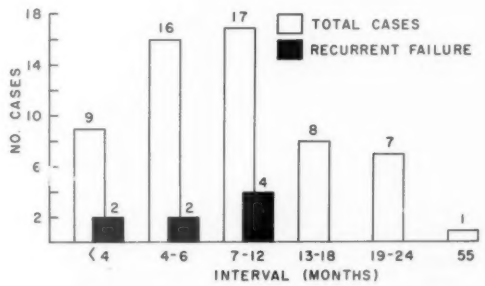


Fig. 16.—Duration of function in 58 successful cases of femoropopliteal regraft.

The results of reoperation have been entirely satisfactory. A normal pulsatile blood flow was restored in 58 of the 64 patients reoperated on (Table VIII). Death occurred from myocardial infarction in one patient and circulation was not restored in 5 patients because of the extensive nature of the occlusive process, in 4 because of atherosclerosis, and in one because of organized thrombus. Amputation was required in one of these. The peripheral circulation and symptoms of arterial insufficiency remained essentially unchanged from those before the original operation in the remaining patients in whom circulation was not restored. These patients have been followed since the second operation for periods up to 56 months, and success has been maintained in 50, including the first patient submitted to reoperation (Figs. 16-17). A third operation

has been performed successfully in 5 of the 8 patients who had late failure of the second operation; consequently, 55 of these patients (94 per cent) continue to have normal peripheral circulation.

DISCUSSION

The results of this study emphasize the usefulness of reconstructive operation in the treatment of patients with atherosclerotic occlusive disease of the lower extremities. Obstructing lesions of the distal aorta and iliac arteries are practically always segmental with a patent distal arterial bed; consequently, lesions at this level are always amenable to operation. By use of one of a variety of procedures depending upon the nature and extent of the lesion, distal circulation can be successfully improved in 96 per cent of the cases. Long-term follow-up, more than 6 years in some instances, indicates that these good results persist indefinitely in most patients; recurrent difficulties have developed in only 6.2 per cent of our cases. Particularly noteworthy is the fact that circulation was successfully restored by reoperation in 33 of the 38 patients who had recurrent difficulties after the first operation. Thus, improvement in circulation has been maintained to date, or until the patient died, in 96 per cent of our cases.

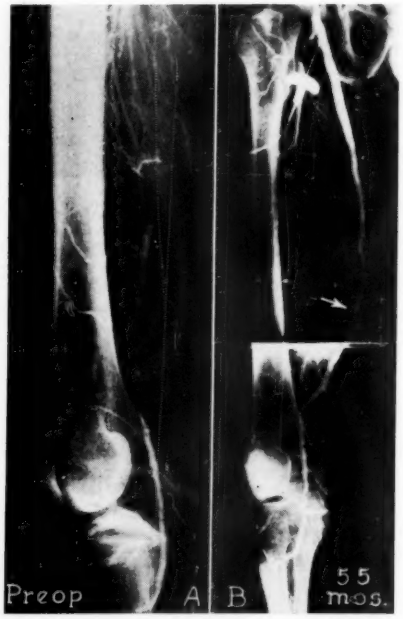


Fig. 17.—Illustrations of first patient reoperated on. *A*, Arteriogram made after recurrent occlusion showing obstruction of femoral artery. *B*, Recent arteriogram made 55 months after second operation employing arterial homograft. Irregularity of graft (*arrow*) was thought caused by deformity produced by suturing several segments of varying sizes to form long graft, although aneurysm cannot be excluded.

Atherosclerotic occlusive lesions of the femoral and popliteal arteries, although frequently extensive, are segmental with a sizable patent channel proximal and distal to the lesion in more than 60 per cent of the cases. By use of endarterectomy for discrete lesions limited to a short segment of artery or the bypass graft using a flexible knitted Dacron tube for more extensive lesions, a distal pulsatile blood flow can be restored immediately in 90 per cent and

maintained in 75 per cent of the cases for the duration of the follow-up extending more than 2 years in many instances. This immediate restoration of circulation results in relief of symptoms in all patients and, particularly, prevents amputation in patients with ischemic cutaneous lesions. These objectives of therapy unfortunately cannot be obtained with any other method at present, and despite the fact that subsequent difficulties develop in a number of patients with femoropopliteal occlusion, conservation of collateral channels by employing the bypass procedure has prevented progression of symptoms in most patients. In fact, in a considerable number of patients a significant degree of improvement occurs after a temporary period of graft function. This feature of temporary graft function assumes great importance in patients with indolent cutaneous lesions precipitated by infection and trauma. A brief period of supplemented arterial blood flow is frequently sufficient to produce healing which persists after graft failure. Moreover, in most patients with recurrent difficulty the lesion remains segmental and amenable to reoperation. By performance of a second operation, good peripheral circulation can be restored in most instances.

The late failure problem has been distorted by the results obtained from earlier procedures and graft materials that have subsequently proved of limited value. Conclusions based upon these observations alone would be discouraging; however, with increasing surgical experience and better understanding of the disease, both the immediate and long-term functional results have continued to improve. Review of our own experience indicates that most late failures were due to technical surgical factors. Technical mishaps, use of graft materials of limited function and application, and disregard for certain manifestations of the occlusive process caused most complications that resulted in failure. Progression or recurrence of the underlying disease was rarely the primary cause of failure. This analysis of the problem, suggesting that most failures are preventable, offers the opportunity to improve the results of reconstructive operation by improvements in surgical technique, better graft replacements, improved methods of arterial visualization, and more thorough understanding of the disease.[4]

Finally, responsibility must be assumed for the many patients in whom reconstructive operation has already been employed. As previously indicated, a variety of complications do occur which occasionally may pose a threat to both the patient's life or extremities. Fortunately, these complications are rare, but a defeatist attitude may be easily assumed in cases involving such complications. However, as this study has shown, by aggressive action a successful result may be obtained in most instances. For example, aortoduodenal fistula must be suspected in every patient with upper gastrointestinal bleeding after abdominal aortic graft operation. Immediate operation is indicated because the diagnosis cannot be established and proper treatment instituted by any other method. Likewise, immediate reoperation is indicated in patients whose symptoms of arterial insufficiency are worse after graft closure, particularly if loss of the extremity is threatened. As previously indicated this situation usually results from obliteration of the distal central channels by a thrombus which has extended from obstruction in the graft or more proximal disease. This thrombus can be removed when operation is performed soon after graft closure, and a normal pulsatile blood flow restored by insertion of a new graft.

SUMMARY

Although a high degree of success may be obtained initially in patients having reconstructive operations for atherosclerotic occlusive lesions of the aorta and the iliac, femoral, and popliteal arteries, continued observation reveals a significant number of recurrent difficulties. A study was made of this problem in the treatment of 1,225 patients in order to evaluate this form of therapy, to discover possible causative factors that might be eliminated or minimized by improvement in technique, and to evaluate the methods of treatment of patients with recurrent occlusion.

By means of a variety of procedures adapted to the nature and extent of the occlusive process, peripheral pulsatile circulation was initially restored in 96 per cent of the 638 patients treated surgically for lesions of the aorta and iliac arteries. Some of these patients were followed for periods longer than 6 years. Thirty-eight patients (6.2 per cent) had recurrent difficulties resulting from aneurysmal dilatation of the graft, false aneurysm, aortoduodenal fistula, or recurrent obstruction. These complications are preventable with present knowledge of treatment of lesions in this location. Particularly noteworthy is the fact that circulation was successfully restored in 33 of the 38 patients who had a second reconstructive operation.

Peripheral pulsatile circulation was restored in 88 per cent of the 587 patients on whom reconstructive operations were performed for occlusion of the femoral and popliteal arteries. The best results (91 per cent) were obtained with the bypass procedure and a flexible, knitted Dacron graft. This procedure was used in more than 50 per cent of the cases. The poorest results were obtained in the small number of patients treated early in our experience by endarterectomy or excision and graft replacement. Late failure occurred in 109 patients (21 per cent) and varied with the type of procedure and graft replacement employed. Failure occurred in most patients who had excision and graft replacement. Long-term functional results of the flexible, knitted Dacron tube inserted as a bypass graft were superior to those in which braided nylon or homografts were used. Most of these failures resulted from preventable complications, and circulation was restored in 58 of 64 of these patients (91 per cent) by reoperation. This study indicates that the immediate and long-term functional results of operation may be significantly improved by increasing surgical experience, better understanding of the underlying disease, improved methods of arterial visualization, and better graft material. Also aggressive treatment of late failures is usually successful.

REFERENCES

1. Cannon, J. A., Barker, W. F., and Kawakami, I. G.: Femoral Popliteal Endarterectomy in Treatment of Obliterative Atherosclerotic Disease, SURGERY 43: 76-93, 1958.
2. Crawford, E. S., De Bakey, M. E., and Cooley, D. A.: Clinical Use of Synthetic Arterial Substitutes in Three Hundred Seventeen Patients, A. M. A. Arch. Surg. 76: 261-270, 1958.
3. De Bakey, M. E., Crawford, E. S., Cooley, D. A., and Morris, G. C., Jr.: Surgical Considerations of Occlusive Disease of the Abdominal Aorta and Iliac and Femoral Arteries: Analysis of 803 Cases, Ann. Surg. 148: 306-324, 1958.

4. dos Santos, J. C.: Sur la désobstruction des thromboses artérielles anciennes, Mem. Acad. chir. **73**: 409-411, 1947.
5. Szilagyi, D. E., Whitcomb, J. G., and Smith, R. F.: Causes of Late Failures in Grafting Therapy of Peripheral Occlusive Arterial Disease, Ann. Surg. **144**: 611-634, 1956.
6. Warren, R., and Villavicencio, J. L.: Iliofemoropopliteal Arterial Reconstructions for Arteriosclerosis Obliterans: Factors Influencing Late Patency, New England J. Med. **260**: 255-263, 1959.
7. Wylie, E. J., and Goldman, L.: The Role of Aortography in the Determination of Operability in Arteriosclerosis of the Lower Extremities, Ann. Surg. **148**: 325-342, 1958.

Discussion

(Papers by Key, Bigelow, and Farber; Crawford, De Bakey, Morris, and Garrett)

DR. SHEPHERD (Boston, Mass.).—I would like to stress the importance of long-term follow-up in tracing the progress of these patients postoperatively.

We have operated on a group of 40 patients with aortic occlusion over a period of greater than 15 months. The 40 patients are divided into two groups: 14 had a thromboendarterectomy and 26 were treated by insertion of grafts, usually bypass gafts. Excluding the 9 deaths or closures in the hospital (one of which was a patient lost to follow-up), there were 31 patients available for follow-up. The long-term patency rate is 64.5 per cent in the whole series, not as high a percentage as has been reported by many other groups.

The thromboendarterectomies were performed chiefly in the high localized occlusions. The grafts were used in the more extensive occlusions, and we feel that this accounts for the difference in results between the two types of surgery.

One of the major factors which Dr. Key mentioned, and we also feel is very important in the incidence of closure, is the appearance of lower occlusion in the femoral arteries below the aortoiliac level. In this series, we have treated 11 patients by long grafting; these grafts are carried from the aorta or iliac artery down to the low femoral or popliteal artery (this is a bypass procedure). Nine of these patients had a successful result, and they are all symptomless.

Twelve other patients who had evidence of femoral disease were not treated in this way, and the results in this part of the series were not so good in that a number of them had residual symptoms and the incidence of closure was much greater. The 9 closures in this group all had some evidence of femoral disease.

In patients with occlusions with a high aortoiliac level, and also a low femoral popliteal level, operative treatment alone is not likely to produce good results, and early closure of the reconstruction is likely. In these cases, it is possible that the procedure of choice is a long bypass graft carried from the aorta or common iliac past both occlusions down to the popliteal artery.

DR. EDWARD J. WYLIE (San Francisco, Calif.).—We share Dr. Crawford's interest in determining the cause of failure following arterial reconstruction. For this purpose we recently reviewed our series of 230 patients who had undergone thromboendarterectomy for what were thought to be purely segmental occlusive lesions at the aorta-common iliac level. Over a 9-year period there were 7 instances in which reocclusion occurred in the endarterectomized segment. All these lesions were re-explored. Recurrence of frank atherosclerosis in the endarterectomized vessel was observed in only 2 cases. In the other 5, atheromatous occlusion had developed distal to the lower level of endarterectomy. Subsequent proximal propagation of intraluminal thrombosis resulted in occlusion of the previously endarterectomized segment.

(Slide.) This preoperative aortogram shows what appears at first glance to be an incompletely occlusive lesion in the proximal one-third of the right common iliac artery. Endarterectomy was confined to the involved segment, leaving behind the slightly thickened intima which can be seen at the common iliac bifurcation. Two years later, intimal thickening at the lower level had progressed to complete occlusion and required reoperation.

(Slide.) This aortogram shows segmental occlusion of the right common iliac artery. Operation was limited to this segment. We were reluctant to extend the operation to the opposite side, since only a minimal lesion could be palpated in the proximal left external iliac artery. The lesion was a narrow tongue of atheroma on the posterior wall, a common lesion rarely visible by aortography. Within 4 years, the tongue had progressed to the extent that it caused complete left external iliac occlusion.

As a result of these and similar experiences in 3 other cases, we have concluded that long-term arterial patency following aortoiliac thromboendarterectomy can be assured only by removal of all zones of thickened intima adjacent to occlusive lesions.

(Slides.) These two slides indicate the rate of growth of an atheromatous deposit in one patient during a 2-year period. This man originally underwent successful popliteal thromboendarterectomy for the 3 cm. lesion shown on the aortogram. An asymptomatic lesion in the left common iliac artery, with an approximately 20 per cent narrowing of the arterial lumen, was shown on the same study. Two years later, aortography showed that the iliac constriction had progressed to almost complete occlusion, resulting in claudication of the left thigh and weakening of the left common femoral pulse.

Dr. Crawford's approach to the investigation of late failures, including repeated arteriography and re-exploration, is particularly vital if we are to remedy the causes of failure.

DR. ROBERT R. LINTON (Brookline, Mass.).—I have been interested in the subject of arterial reconstructive surgery by the utilization of various types of grafts for a number of years. I was somewhat surprised to find that from the report fom Toronto, we only heard of one autogenous vein graft that was used, despite the work done by Dr. Gordon Murray many years ago on the success of various types of autogenous venous grafts.

There still seems to be a need for the ideal graft, and I would like to complement what Dr. Lord has already said, namely, that the saphenous vein is the best graft that we now have available for occlusive disease in the lower extremity. I would like to document this for you with a few colored slides and arteriograms in a man in his early 50's with a block in his superficial femoral artery. In 1956, I implanted an arterial homograft from the common femoral artery down to his popliteal artery. (Slide) Aneurysmal dilatation of the homograft developed, as you see in this slide, which was taken 2 years after the grafting procedure.

Because I did not have sufficient homograft at that time, I had to use a small piece of saphenous vein autograft to make the bypass graft sufficiently long. This was implanted at the distal end. The slide shows a distal anastomosis between the homograft here and the saphenous vein autograft, and also the popliteal artery where it was implanted. You will notice that the saphenous vein looks about the same size as the day I put it in.

(Slide.) One year later, the proximal part of the homograft had become aneurysmal in this short period of time. This is one of the great difficulties and faults I have found with homografts and the reason I hesitate to use them any more for femoral grafts. If you will remember what this homograft looked like one year before and compare it with this picture, you will see what tremendous degenerative changes have taken place in the last 12 months of the third year in which the graft had been functioning.

(Slide.) When I took out the aneurysmal, dilated homograft at the second operation, I inserted another piece of saphenous vein autograft and here is the original saphenous vein which had been functioning for 3 years, and the one which I had put in one year previously. Neither has dilated—both look exactly the same size as the day they were implanted.

(Slide.) This shows the aneurysmal dilatation of the homograft, and I would like to again demonstrate the utilization of autogenous tissue in this case, not only by the use of autogenous venous grafts, but also by means of endarterectomy popularized by Drs. Cannon and Wylie. It was possible, after endarterectomizing the common femoral and

superficial femoral arteries, to anastomose the proximal end of the saphenous vein grafts, which I had already implanted and were in place, to the distal end of the endarterectomized superficial femoral artery with complete restoration of circulation of the extremity, and leaving the patient now with entirely autogenous tissue. In every instance, if you can use autogenous tissue either by utilization of saphenous autograft or endarterectomizing the femoral artery, these are the preferable procedures, and my first choice is the former.

DR. JOSEPH M. JANES (Rochester, Minn.).—I am not from Toronto, but I would like to come to the defense of Toronto. In the early 1930's, Dr. Gordon Murray, a senior member of this organization, was inserting autogenous vein grafts for popliteal aneurysms.

DR. OSCAR CREECH, JR. (New Orleans, La.).—We spend a great deal of time talking about runoff area, and perhaps it is the runin which is of equal if not more importance. I think Jack Wylie has also emphasized this in the past. We have had a number of patients with complete occlusion of the femoropopliteal system in whom it was impossible to establish a good runoff area in the sense that the anterior and posterior tibials were open, but, by opening the superficial femoral and popliteal arteries down to the bifurcation, we were able to maintain a good flow to that area, through collaterals coming off laterally from the superficial femoral.

In patients who have had femoral bypass grafts that became occluded, we have used thromboendarterectomies in several instances and have obtained a successful restoration of blood flow.

(Slide.) This is an example of a bypass graft in a patient who had superficial femoral obstruction which subsequently became occluded. This patient had inflow occlusion as well as outflow occlusion. We performed a thromboendarterectomy both above and below, and, by doing this and leaving the graft in place, we were able to restore pedal pulses and relieve symptoms. This has been done in several instances and seems to be a satisfactory approach to the problem of graft failures.

DR. JERE W. LORD, JR. (New York, N. Y.).—I would like to describe one patient with obstruction from a different cause. He was an individual of 35 who presented with gangrene of the toes of one foot and markedly diminished circulation for 4 months. An aortogram (slide) made in January, 1957, shows stenosis at the origin of each of the common iliac arteries with thrombotic narrowing of the distal aorta. He was treated by thromb-endarterectomy as Dr. Wylie outlined and taught me. His pulses were excellent, his gangrene demarcated, and his toes were saved. Oscillometrics went from near 0 to around 10 in each leg.

He did very well for 6 months. Then, rather abruptly he developed claudication again. In October, 1957, some 8 months after the first operation and after 6 months with good results, he was explored. It was anticipated that either disease had been overlooked, which I did not think likely, or more probably that he had new arteriosclerotic occlusive disease. The aortogram done before the second operation suggested arteriosclerotic narrowing in the terminal aorta at the beginning of the common iliac. At operation we found what I believe to be a fibrotic periarterial squeezing of the common iliac arteries and of the terminal aorta. Fear of causing a dangerous hemorrhage from the inferior vena cava and iliac veins made me decide to divide the aorta above and the common iliacs beyond the point of narrowing. We opened the obstruction and found a perfectly smooth intima without any sign of thrombosis or any sign of the intimal placquing. I believe, therefore, that he had periarterial fibrosis, although following his first operation he had a perfectly benign clinical course. This time we inserted end-to-end a Dacron prosthesis, one of Drs. Julian and Deterling's material, a product of the Ethicon Company, with end-to-end aorta above and end-to-end common iliacs below.

He did well for 10 months, had excellent pulsations on limited walking distance, and then began to show signs of shutting off.

Because this was a sturdy young man and because of his remarkable faith in his professional care in spite of two surgical onslaughts, last December, 6 months ago, he

underwent a third operation, and this time I had the opportunity to use one of Dr. De Bakey's crimped Dacron prostheses, and he had again an end-to-end above, end-to-end below on the right of the common iliac, and end-to-side to the external iliac on the left.

He was in the office on Friday, now has excellent pulsations, excellent oscillometrics, but he has still not gone the critical year, and I am hoping that Dr. De Bakey's material will be more satisfactory than that previously used.

DR. KEY (closing).—Dr. Linton will be glad to know that we are using some autogenous vein grafts now, and I was very glad that Dr. Janes paid a tribute to Dr. Murray of Toronoto. I now understand that he has some patients that he has followed for as long as 18 to 20 years after the use of vein grafts.

The paper was not in any way an attempt to compare the newer materials to the older. We do not use nylon now and we use very few homografts. The idea was, as I said, to give us a yardstick for future comparison of such materials.

In the long-term results with these grafts, we are going to have inevitable failure either of the graft or the patient's heart. Dr. Spaulding in a review of patients from the onset of claudication noted in the age group 55 to 65, there was a 4 per cent mortality in 10 years. It would seem, therefore, perhaps in some of these patients, we are not only restoring the life of the limb, but we appear to be increasing the longevity of the patient.

CHANGES IN THE PERIPHERAL PULSES AFTER SYMPATHECTOMY FOR ARTERIOSCLEROSIS

UGO RUBERTI, M.D., EDWARD A. EDWARDS, M.D., AND LESLIE OTTINGER, B.A., BOSTON, MASS.

(From the Department of Surgery, Peter Bent Brigham Hospital, and the Department of Anatomy, Harvard Medical School, Boston.)

THE absence of a palpable pulse constitutes the most direct evidence of obstruction of the artery to that part. Conversely, the reappearance of the pulse is a certain sign that arterial patency has been restored. This simple physical sign is all we need to tell us that reconstruction of an arteriosclerotic vessel has been accomplished, and that prior ischemic changes will be partly or wholly overcome. The pulse as palpated by the examiner's finger has been of only minor utility in following the course after sympathectomy. Only uncommonly, and then usually after some weeks, has a previously absent pedal pulse become palpable after this operation. It is nevertheless known that the pulsation in the arterial tree beyond an obstruction is not wholly damped; it may be expected that a sensitive device can demonstrate pulsation where the finger does not.

We have indeed found that such an apparatus, consisting of a pneumatic cuff and an electronic transducer of differential capacitor type, records a pulse trace from all levels of the limb, including the toes, in the majority of patients with arteriosclerosis. The pulse record is moreover subject to quantitative evaluation, so that changes from one occasion to another may be stated in numerical terms.[7]

We present here the results of such study of the pulses recorded from several levels of 35 arteriosclerotic limbs in 26 patients examined before and after lumbar sympathectomy, and followed 6 to 27 months after operation.

MATERIAL AND METHOD

Patients about to undergo sympathectomy were entered consecutively in the study as laboratory time was available. Since our pool of patients had already been culled for those suitable for reconstructive operation, those chosen for sympathectomy included more examples of diffuse and distal disease than a completely unselected group. Preoperative study included aortography or femoral arteriography in many instances. In occasional cases, the pulses of the foot and toe were measured preoperatively before and after an attempt to produce reactive hyperemia through the application of a constricting cuff at the ankle.[8]

Thirty-four patients were originally thus enrolled, but 2 were dropped because reconstruction and sympathectomy had been done simultaneously; 2 died within 6 months; and 4 were lost to follow-up. The 26 patients presented were followed 6 to 27 months after

Aided by Grant No. H-2661 from the National Institutes of Health of the U. S. Public Health Service.

Read at the Thirteenth Annual Meeting of the Society for Vascular Surgery, Atlantic City, N. J., June 7, 1959.

operation, with an average of 12.3 months. The postoperative examinations averaged 5 in number. There were 17 men and 9 women. Four men and 2 women were diabetic. The patients' ages varied from 51 to 80 with an average of 65 years.

The operation was performed through an anterolateral extraperitoneal approach, with removal of that part of the chain overlying the spine from either the top or lower edge of the body of the second lumbar vertebra, down to the fourth disc. Such a removal included the second, third, and fourth lumbar segments almost completely, and also some or all of the first segments when the removal ran to the upper edge of the second vertebra.[3] The adequacy of denervation was checked by skin resistance measurement in every instance. High electrical resistance was demonstrated at least as far proximal as the knee, and in some patients to the groin.

Only the main features of the pulse registration need be mentioned here, since a detailed description is available elsewhere.[7] The limb is examined at several levels from great toe to lower thigh. A pneumatic cuff of proper size is placed around the level to be examoined. It is connected to the pneumatic outlet of the transducing unit, and inflated to 30 mm. Hg. The transducer is a differential capacitor, the electrical output of which is fed into a direct writing recorder. A paper speed of 25 mm. per second is used throughout.

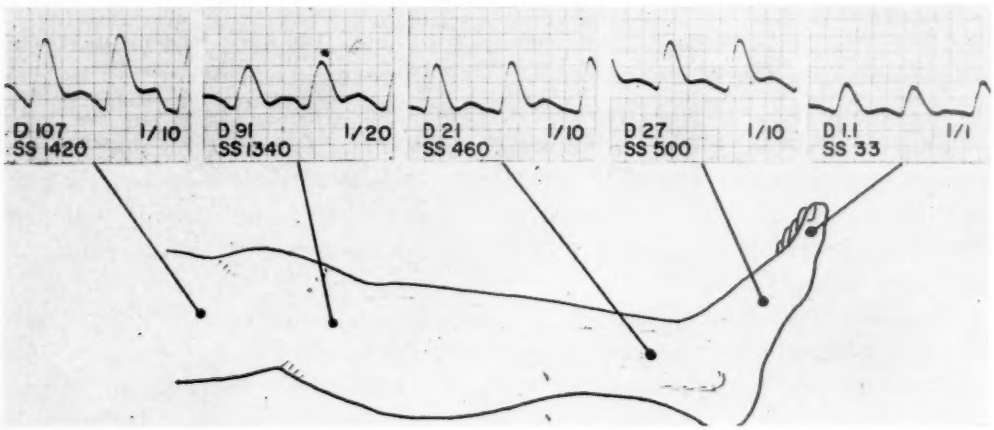


Fig. 1.—Normal pulses of the lower extremity of a large strong man 22 years of age. (From Edwards and Ottinger: New England J. Med. 259: 101, 1958.)

A device in the apparatus gives a calibration of the pen deflection for each cuff application. With this, plus a planimetric examination of the area under the pulse trace, one is able to determine 2 numerical attributes of the pulse: mean pulse deflection (mean change in volume in cubic millimeters, during 1 pulse cycle), noted as *D* (Fig. 1); and systolic slope (rate of systolic increase in volume, in cubic millimeters per second), noted as *SS* (Fig. 1). The fraction shown in some figures denotes the attenuation of the recording used in the particular pulse trace shown. The changes in the configuration of the pulse imposed by arteriosclerotic narrowing or obstruction may be seen in Fig. 2. Both deflection and systolic slope are reduced, the dicrotic notch is flattened or lost, and aberrant vibrations are noted, particularly in the diastolic limb.

As the study progressed it was apparent that changes in systolic slope roughly paralleled those in mean pulse deflection. We are therefore focusing in this presentation on the deflection alone.

The actual pulse tracings are shown for certain selected cases. In order to present the data concisely for each limb, however, all the postoperative values for the deflection at each site have been averaged, and this average compared with the preoperative level at the same site of that limb. This comparison is expressed as percentage of increase or decrease from the preoperative level. Where the preoperative pulse trace of the toe was hardly discernible and the planimetric measurement necessary to computation impossible, a minimal D value of 0.1 was ascribed to the curve and used for the later comparisons.

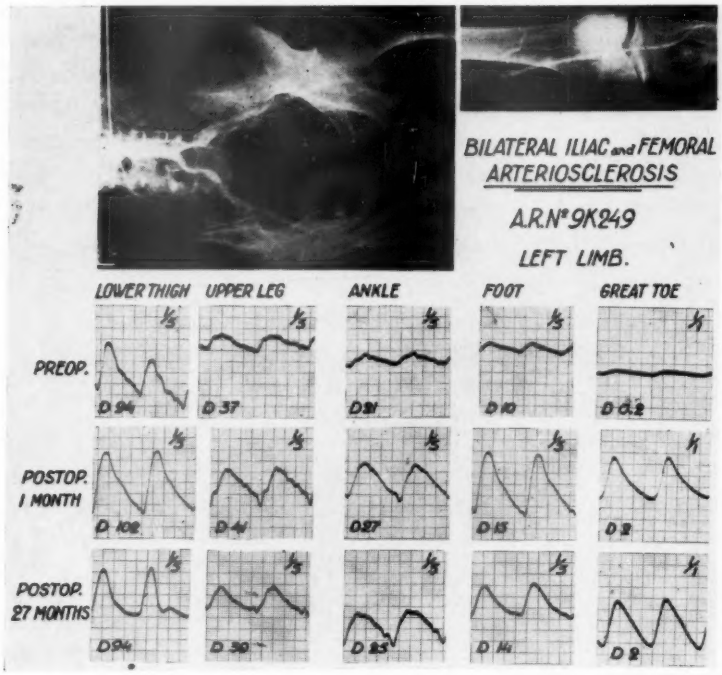


Fig. 2.—Pulse and radiographic study of a patient with bilateral iliac narrowing, and occlusion of both superficial femoral arteries. The postoperative averages for pulse deflection placed the case in category A (Case 7). Both posterior tibial pulses became palpable 13 months after operation. Claudication was significantly lessened.

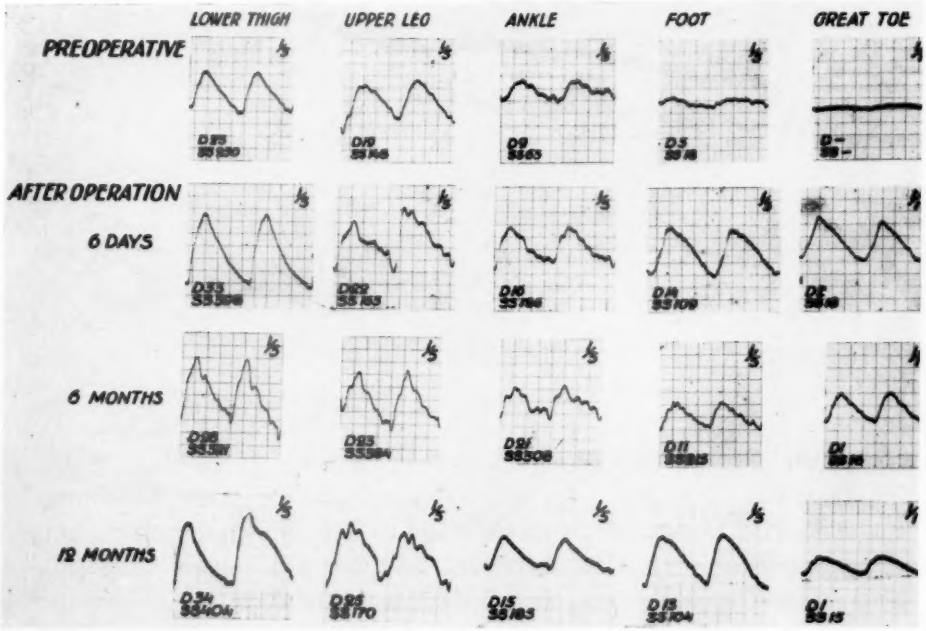


Fig. 3.—Increases of pulse deflection at all levels in Case 9 in category A. At 19 months the values for pulse deflection were: thigh, 52; leg, 39; ankle, 20; foot, 17; great toe, 2. The patient was a 65-year-old woman, with a painful cyanotic foot. No pulse was palpable below the femoral. The oscillometric reading (Pachon) at the calf was 0.5 unit at 110 mm. Hg, and zero at the ankle. The dorsalis pedis became palpable 6 months after operation. (From Edwards, Ruberti, and Ottinger: *in* Peripheral Vascular Diseases, Philadelphia, The Blakiston Company. In press.)

RESULTS

We have attempted to assemble our cases into groups of similar response to facilitate comparison with clinical data. The patients fell into 3 categories: those with increase in pulse deflection at all levels of the limb, noted as category A, 12 limbs; those with increase in deflection mainly in the distal parts of the limb and usually a fall proximally, noted as category B, 9 limbs; last, those with a decrease at most levels, noted as category C, 14 limbs. Only one patient (Case 5) is represented in two categories, 1 limb in A, the other in B.

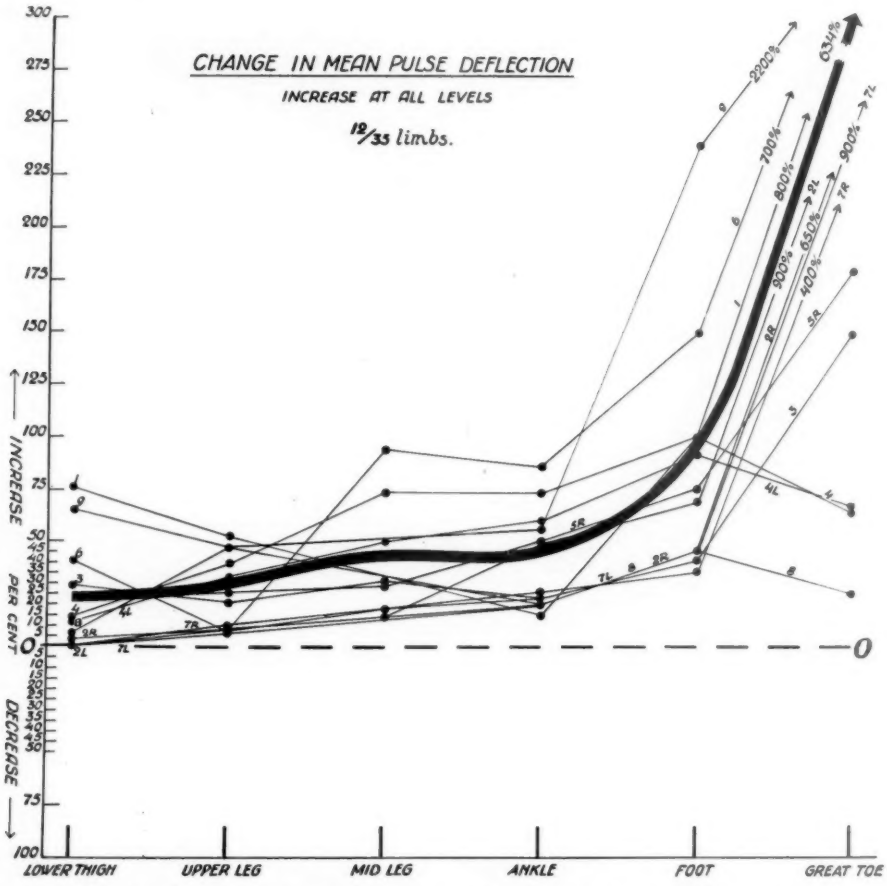


Fig. 4.—Individual pulse responses and average of category A. Numbers refer to case and limb, right (*R*) or left (*L*).

Improvement, as seen in category A (Fig. 4), was extreme in the toe, falling to a moderate level in the foot and to quite modest levels more proximally. The chronology of improvement was similar to that known for skin temperature after sympathectomy; that is, there was maximal increase in deflection in the first few days after operation, then a vacillation of effect, with stability, close to the maximum, reached in some weeks and maintained thereafter (Figs. 2 and 3). In some limbs pulse increase was at first limited to the toe and foot, with ascent of effect proximally in the succeeding weeks or months. The increase in pulse deflection is accompanied by a general tendency toward a more normal configuration of the pulse (Fig. 2).

In category B (Fig. 5) a decrease in the deflection was usually noted from the mid leg proximally, with a fair to good increase in the more distal parts. The degree of distal increase in deflection varied considerably from case to case, but the average was noticeably lower at the toe than in category A. In regard to the sequence of change, cases in category B were more likely to show early increases limited to the toe, with slow increase over some months, at the foot and ankle, and with concomitant decline in the proximal levels.

With minor exceptions, the pulse deflection decreased at all levels in category C (Fig. 6). Only one limb showed a substantial increase in the toe. All cases showed a rather steady lowering or essentially unchanged pulse status. There were no instances of initial improvement followed by later deterioration.

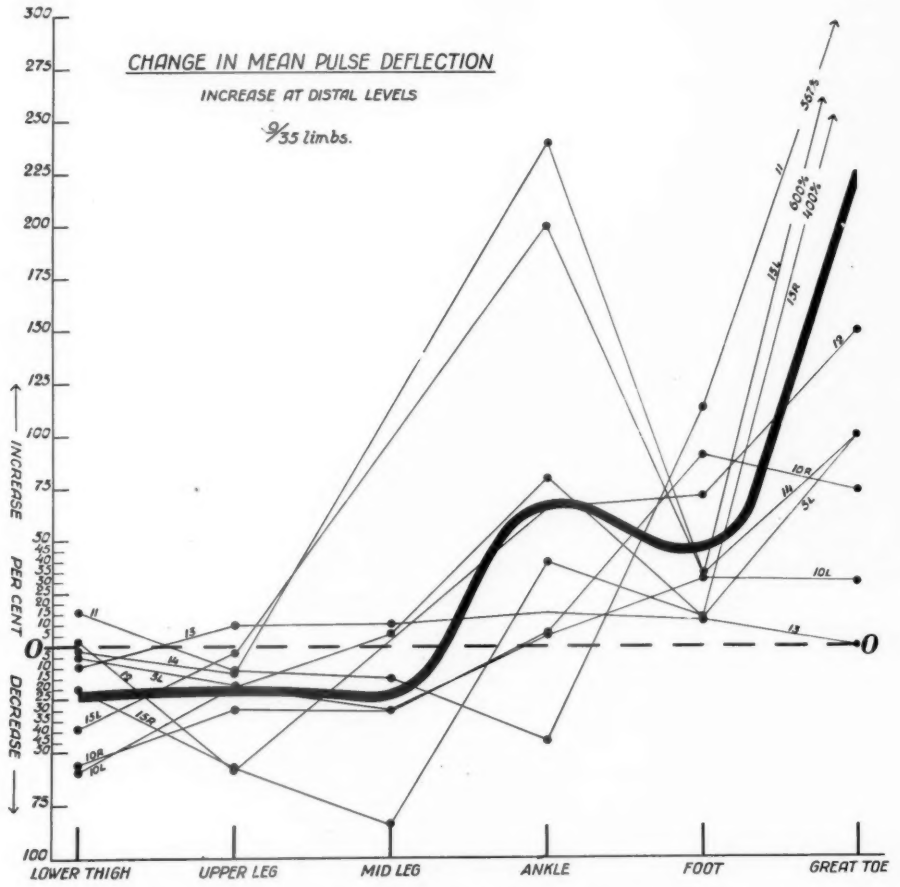


Fig. 5.—Individual pulse responses and average of category B.

Correlation With Clinical Status.—Table I shows the levels of the lowest palpable pulse, and Table II summarizes several clinical attributes which appear significant for the patients and limbs of each of the designated categories. We have omitted the data on age, sex, and exact level of postoperative skin resistance change, since these factors showed no particular trend.

As a group, the patients in category A showed little evidence of general disease—the ratio of pedal ischemia to claudication as the presenting problem

TABLE I. LEVEL OF LOWEST PALPABLE PULSE (NUMBERS REFER TO LIMBS)

	CATEGORY A 12 LIMBS—9 PATIENTS	CATEGORY B 9 LIMBS—7 PATIENTS	CATEGORY C 14 LIMBS—11 PATIENTS
Aortic	0	0	4
Femoral	7	5	8
Popliteal	3	1	1
Pedal	2	3	1

TABLE II. CLINICAL ATTRIBUTES OF PATIENTS (NUMBERS REFER TO LIMBS)

	CATEGORY A 12 LIMBS—9 PATIENTS	CATEGORY B 9 LIMBS—7 PATIENTS	CATEGORY C 14 LIMBS—11 PATIENTS
General status			
Diabetes	2	0	4
Severe cardiac or central nervous system disease	0	0	3
Preoperative limb status			
Claudication	4	3	2
Pedal ischemia	8	6	12
Prior occlusion femoropopliteal reconstruction	0	1	3
Level of occlusion (Table I)	Generally low; no aortic or iliac		Some aortic or iliac
Oscillometry (Pachon)	Generally no lower than ½ unit: no zero readings at calf		Many zero readings at calf and ankle
Results			
Reappearance of palpable pedal pulse	4	1	0
Walking ability	Generally increased		5—continuous foot pain on activity
Amputations	0	0	1 (thigh)

was low (2 to 1); none of the patients had experienced occlusion of a reconstructed artery; and pulse amplitude, measured by oscillometry, while sometimes low, was never at the zero level in the calf and rarely at the ankle. The clinical response in these patients was good: ischemic signs in the foot were overcome; there was improved walking ability, both in regard to lessened claudication and the loss of foot discomfort; finally, some pedal pulses became palpable in 5 weeks to 7 months.

Patients in category C showed more evidence of general disease. There was a higher ratio of pedal ischemia to claudication (6 to 1); claudication distance was measured in feet, rather than in yards or blocks as in the first category; and the foot problems included more examples of superficial ulceration. There were 4 instances of complete, and one of incomplete, occlusion at iliac level. Prior occlusion of a reconstructed femoropopliteal artery had occurred in 3 limbs. The oscillometric reading was zero at the ankle, or ankle and calf, in several. Postoperatively, all patients showed some improvement in foot color

and temperature, but it was minimal. In none did a pedal pulse become palpable. Continued ulceration in one patient led to an unsuccessful transmetatarsal, then to supracondylar, amputation. In 5 limbs, preoperative rest pain disappeared; but the patients could do but little walking without bringing on pain in the foot.

The patients and limbs of category B appeared midway between the other 2 categories with respect to their preoperative state and postoperative result. In this group occurred the only instance of postoperative loss of a pulse, that of the popliteal in Limb 5L.

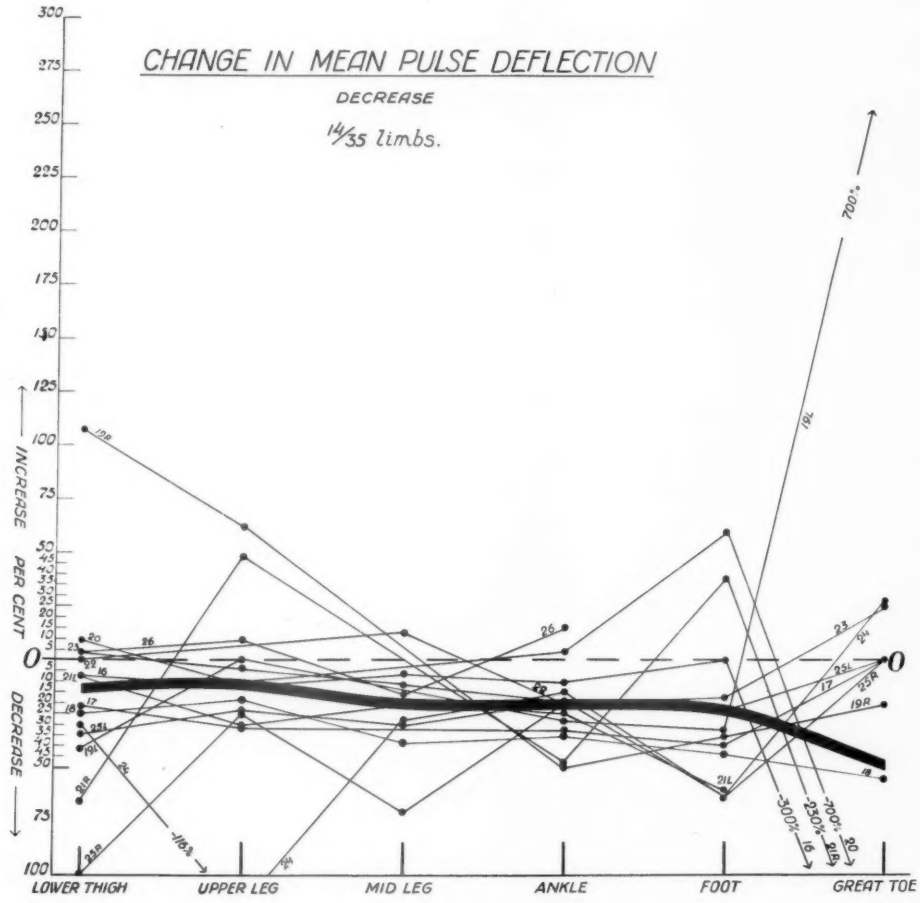


Fig. 6.—Individual pulse responses and average of category C.

While this classification shows a general conformity of clinical attributes and behavior of each group with the postoperative pulse response, it must be emphasized that there were individual exceptions. It is therefore invalid to attempt a certain prediction of outcome of sympathectomy in any one patient on the basis of the clinical classification suggested. An ability to induce reactive hyperemia preoperatively[8] was followed by a good response, but a fair response sometimes followed a poor reactive hyperemia test.

Effect on a Previously Denervated Contralateral Extremity.—In 4 patients sympathectomy of the contralateral limb had been performed one or more years prior to the presently studied operation. We were interested to see what effect the new operation might have on the side previously operated upon, where the result of its homolateral operation was now well stabilized. It has been suggested that a second operation might improve the first by removing the source of crossed sympathetic communications. We found no such increase. A decline in pulse deflection was noted during the first week, but this is probably not significant since this is a nonspecific effect of any operation. Later the pulse showed no sustained variation from its preoperative level.

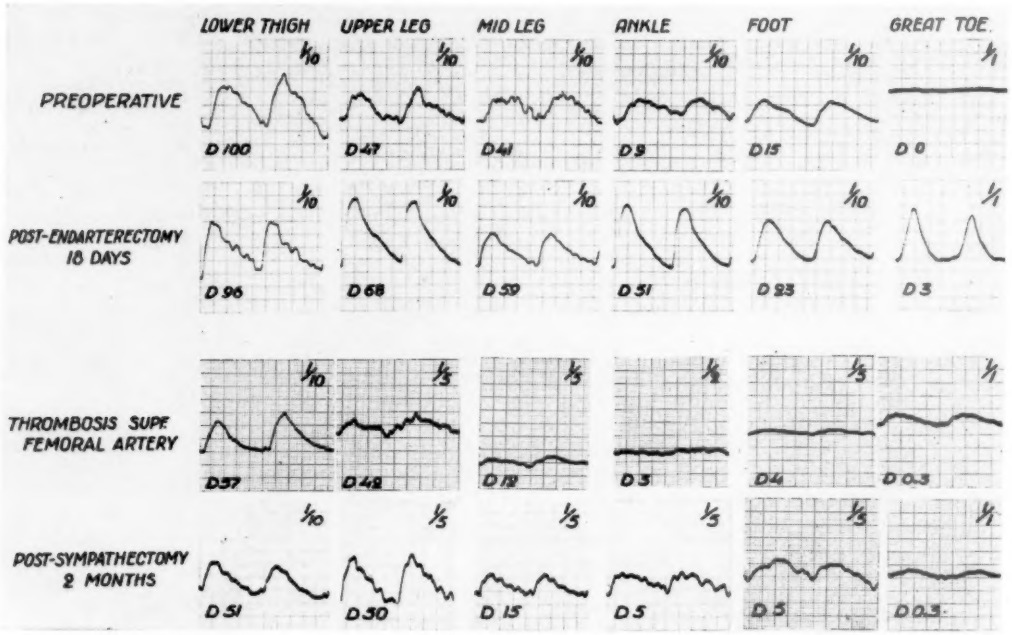


Fig. 7.—Pulse changes after superficial femoral endarterectomy, followed by thrombosis of this artery, and sympathectomy. Case 13 in category B.

DISCUSSION

The increase in pulse amplitude in categories A and B is an indication of a favorable influence of the operation upon the arteries of the limb. As such, this conforms to other observations of the effect of sympathectomy upon the arteries by the techniques of oscillometry,[1] plethysmography,[12, 14] and manometry of the smaller vessels.[10, 11] The effect upon the pulses in the proximal parts of the limb also agrees with the postoperative increase in calf muscle power previously reported from this laboratory.[2] The degree of increase in pulse deflection, and of modification of the abnormal pulse configuration, would seem strong evidence for an influence not only upon cutaneous arteries, but rather upon all the arteries in the cross section of limb under the cuff.

The increase in pulse deflection at the toe compares favorably with that we have seen after reconstruction at aortoiliac or femoropopliteal levels, which

has ranged from 100 to 700 per cent. In contrast to the distal preponderance of pulse increase after sympathectomy, however, that after reconstruction is evenly present over the limb as a whole (Fig. 7).

While the character of a pulse depends upon many factors such as cardiac output, distal resistance, vessel elasticity, and proximity to the surface, progressive increase in deflection in the pulses from the same site, with no new pathology occurring, means dilatation of arteries in that segment of the limb. Moreover, while we cannot compute flow from pulses taken from arteries whose walls are more or less rigid, it may be stated that an increased pulse deflection in this setting means increased flow.

In some cases in category BB, the fall in pulse deflection proximally may not be an unfavorable sign. Through experiment, we have previously shown[7] that distal release of resistance, as exists in those limbs whose distal deflection is increased, diminishes the deflection in the artery proximally, probably through an increased flow in the proximal artery and a fall in its lateral wall pressure.

Considerable interest centers on the cause of the increased deflection at all levels in category A. The enlargement of the distal pulses appears to be due to vasodilation through release of tone, with a magnitude dependent upon freedom from diffuse disease.[4, 9] Certainly many limbs in category C exhibited many of the signs of "diffuse" arteriosclerosis, both by simple observation and by special tests such as arteriography, and the registration of attempted reactive hyperemia.[8] The proximal postoperative dilatation has not been widely appreciated in this country, although it has been adequately demonstrated by arteriography abroad. It is possible that the proximal dilatation is due to release of tone here as well. On the other hand, it may simply be secondary to the distal fall in vascular resistance. A similar relationship exists in arteriovenous fistula, where again there is a great drop in peripheral resistance and consequent increased flow, and, with this, a marked proximal dilatation.

The clinical attributes which correlated with the favorable pulse changes are similar to those we had previously found associated with good clinical effects of the operation.[5, 6]

Our data on the effect of contralateral sympathectomy are hardly conclusive for the presence or absence of functionally significant crossed communications. One deterrent to a potential increase in pulse deflection in the previously operated limb is the tendency of the second operation to cause a shift of blood to the second side.[13]

CONCLUSIONS

1. The peripheral pulses were recorded in 35 arteriosclerotic limbs of 26 patients, followed 6 to 27 months after lumbar sympathectomy. The average follow-up period was 12.3 months.

2. A quantitative comparison was made of the amplitude of the pulses (mean pulse deflection) after operation, with the pulses from the same sites of the individual limb obtained before operation. Changes in systolic slope paralleled those of mean pulse deflection.

3. In 12 limbs there was a postoperative increase in deflection from toe to thigh. While the change was most marked at the toe, foot, and ankle, increases at more proximal levels were sufficient to suggest an influence of the operation upon the entire arterial tree of the limb, deeply as well as superficially. Clinically, these limbs showed obstructions below groin level, with indications of somewhat localized disease. The clinical results were good.

4. In 14 limbs there was a postoperative decrease in pulse deflection at most levels. The arteries were obstructed at iliac levels in some of these limbs, and there were more indications of a diffuse process. The postoperative results were fair to poor.

5. In 9 limbs, there was an increase in pulse deflection from the ankle down. Clinically, these limbs were intermediate in clinical attributes between the other 2 groups.

6. Favorable pulse changes were maintained during the period of observation, except for one instance of postoperative occlusion of the popliteal artery.

7. No sustained variation was encountered in 4 limbs previously sympathectomized when contralateral sympathectomy was performed.

REFERENCES

1. Atlas, L. N.: The Growth of a Collateral Arterial Circulation in the Sympathectomized Arteriosclerotic Leg—an Oscillometric Study, SURGERY **33**: 268, 1953.
2. Edwards, E. A.: Evaluation of Arterial Reconstruction and Sympathectomy by Direct Stimulation Ergometry, A. M. A. Arch. Surg. **76**: 200, 1958.
3. Edwards, E. A.: Operative Anatomy of the Lumbar Sympathetic Chain, Angiology **2**: 184, 1951.
4. Edwards, E. A.: Postamputation Radiographic Evidence for Small Artery Obstruction in Arteriosclerosis, Ann. Surg. **150**: 177, 1959.
5. Edwards, E. A., and Crane, C.: Lumbar Sympathectomy for Arteriosclerosis of the Lower Extremities, New England J. Med. **244**: 199, 1951.
6. Edwards, E. A., and Crane, C.: Lumbar Sympathectomy for Arteriosclerosis. Status of 100 Patients 5 Years After Operation, A. M. A. Arch. Surg. **72**: 32, 1956.
7. Edwards, E. A., and Ottinger, L.: Pulse Registration in the Study of Peripheral Arterial Disease, New England J. Med. **259**: 101, 1958.
8. Edwards, E. A., Ottinger, L., and Ruberti, U.: Pulse Registration as a Means of Evaluating Peripheral Vascular Patency and Vasomotor Activity, Am. J. Cardiol. **4**: 572, 1959.
9. Edwards, E. A., Ruberti, U., and Ottinger, L.: Lumbar Sympathectomy With Special Reference to Arteriosclerosis, *in* Peripheral Vascular Diseases, Philadelphia, The Blakiston Company. In press.
10. Eichna, L. W.: Capillary Blood Pressure in Man. Direct Measurements in the Digits of Patients With Raynaud's Disease and Scleroderma Before and After Sympathectomy, Am. Heart J. **25**: 812, 1943.
11. Gilfillan, R. S., and Corpuz, D.: The Effect of Peripheral Nerve Interruption, Sympathetic Paralysis, and Posture on the Blood Pressure in the Human Extremity, SURGERY **43**: 758, 1958.
12. Messent, D., and Beaconsfield, P.: Circulatory Changes in the Lower Limb Following Sympathectomy, SURGERY **35**: 382, 1954.
13. Occhipinti, S., and Bounous, G.: Modificazioni del flusso arterioso dopo gangliectomia lombare bilaterale nelle arteriopatie obliteranti croniche degli arti inferiori, Minerva Cardioangiol. **3**: 1, 1955.
14. Ruberti, U., and Miani, A.: Modificazioni emodinamiche distrettuali dopo gangliectomia simpatica lombare, Minerva Cardioangiol. **6**: 1, 1958.

SCALENUS ANTICUS SYMPTOMS: EVALUATION AND SURGICAL TREATMENT

DOUGLAS H. RIDDELL, M.D.,* JAMES A. KIRTLEY, JR., M.D.,*
JAMES L. MOORE, M.D., AND ROBERTO S. GODUCO, M.D.,
NASHVILLE, TENN.

(From the Department of Surgery, Vanderbilt University School of Medicine)

IT is evident that the problem of pain in the upper extremity frequently lacks satisfactory explanation. The term "thoracic outlet syndrome" has been used to denote neurovascular compression at the exit of the brachial plexus and subclavian vessels from the isthmus of the root of the neck. Pain, numbness, tingling, vasospasm, and even gangrene have been attributed to neurovascular compression by such structures as a cervical rib, the scalenus anticus muscle, the scalenus medius muscle, a high first rib, anomalous fibrous bands, the subclavius muscle, and costoclavicular compression. Some recent investigators contribute to the growing discord regarding compression of the brachial plexus or subclavian artery by the scalenus anticus muscle alone and others dissent from the idea that the scalenus anticus syndrome without the presence of a cervical rib is a definite factor in upper extremity pain.

As early as 1935 Ochsner and De Bakey[6] stressed that scalenus anticus symptoms could occur when the anterior scalene muscle is hypertrophic or spastic, displacing the neurovascular structures of the thoracic outlet. Symptoms could be produced then by compression of the structures against the first rib without the presence of a cervical rib. Many surgeons have felt that section or excision of the anterior scalene muscle relieved this compression and nothing else was necessary even in the presence of a cervical rib. More recently, however, excision of the cervical rib has been thought to be mandatory if clinical evidence of neurovascular compression in that area is evident. Cervical rib compression and scalenus anticus syndrome give an identical picture. Numbness, pain, and paraesthesia are present in the fingers and hands. Patients are usually of the middle age group. The symptoms are more likely to be along the ulnar distribution of the forearm and hand. Abduction of the arm aggravates symptoms and obliterates the radial pulse, as does also the lifting of a weight, or turning the head to the opposite side. It is generally accepted that many of these people presenting these symptoms without a cervical rib may be treated successfully by conservative methods aimed at relieving muscle spasm and pain. The "costoclavicular syndrome" described in 1943 by Falconer and Weddell[1] produces brachial plexus compression identical with scalenus anticus or cervical rib compression but frequently is accompanied by Raynaud's phenomena. In

Read at the Thirteenth Annual Meeting of the Society for Vascular Surgery, Atlantic City, N. J., June 7, 1959.

*Associate Professor in Clinical Surgery, Vanderbilt University School of Medicine.

some cases of costoclavicular compression venous obstruction is found also and may be demonstrated by venography.

A ruptured nucleus pulposus of the cervical spine may produce symptoms similar to those of scalenus anticus compression. In the former, however, extension of the neck, coughing, or sneezing may accentuate the pain. Arterial or venous compression does not occur with the herniated cervical disc, but spasm of the scalene muscles may be produced secondarily, which further confuses the diagnostic problem. The pain from the herniated cervical disc more frequently radiates to the radial portion of the hand, the shoulder, or the precordium. Cervical osteoarthritis and the superior sulcus syndrome may also produce pain which is easily confused with that of scalenus anticus compression.

The successful treatment of the "thoracic outlet syndrome" depends on the accurate localization of the compression and the exact determination of the abnormal structures involved. Since cervical ribs occur in approximately 0.6 per cent of the population they may be unjustly implicated at times. The occasional presence of aneurysms or thrombosis of the subclavian artery distal to the cervical rib makes its removal almost obligatory even if symptoms are minimal.

This is a review of 53 patients who have been subjected to anterior scalenotomy during the past 15 years for neurovascular compression symptoms of the upper extremity. Most of the patients had an anterior scalenotomy alone while the remaining ones had scalenotomy in conjunction with some other definitive procedure aimed at relieving compression of the thoracic outlet. Seven patients are included who had evidence of venous obstruction in this general area. Four of these with venous obstruction have been reported previously by McCleery and associates.[5]

The 53 patients may be divided into three groups. Group I is composed of 34 patients with evidence of scalenus anticus compression without a cervical rib being present. Group II consists of 12 patients with similar symptoms in whom a cervical rib, unilateral or bilateral, was found. Group III is made up of 7 patients in whom subclavian vein obstruction was the presenting problem with both scalenotomy and subclavius muscle excision being required.

There were 21 males and 32 females with an age range of 18 to 67 years. The mean age was 38.7 years. The left upper extremity was involved in 31 patients while the right was symptomatic in only 15. Seven patients had evidence of bilateral neurovascular compression.

The duration of symptoms extended from 2 weeks to 15 years with a mean duration of 27.5 months. Pain, either radicular or aching in character, was present in 39 patients while numbness and tingling occurred in 36 patients (Fig. 1). Edema of the hand and arm was present in 7 patients. Pain was occasionally present when numbness or tingling was not a factor. Paresthesia on the other hand was present sometimes with an absence of pain. Pain or paresthesia of the hand and fingers occurred in 46 patients while the arm, elbow, or forearm was affected in 41 instances (Fig. 2). Shoulder and subscapular symptoms occurred in 15 patients while neck pain was a complaint in 11 patients.

Objective findings were of great importance in arriving at a decision for operative treatment (Fig. 3). Cervical ribs were visible in the preoperative

roentgenograms in 12 patients with 10 of these having bilateral cervical ribs. Neurological signs were not as common as vascular signs. Some loss of sensation was present in 18 patients. A mass, usually tender, could be felt in the supra-

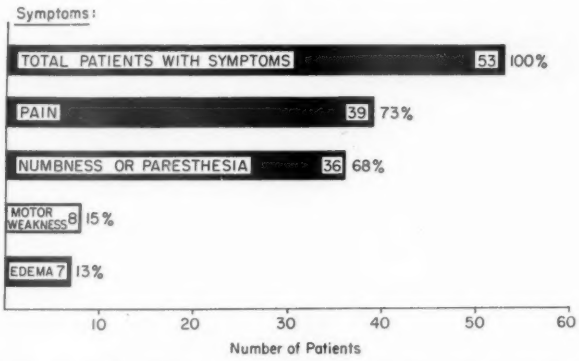


Fig. 1.—Symptoms in 53 patients with thoracic outlet compression.

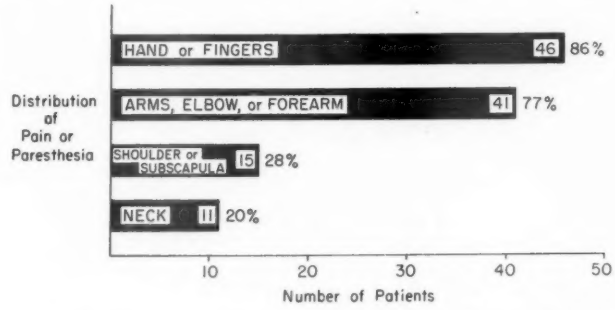

Distribution of Pain or Paresthesia
HAND or FINGERS 46 86%
ARMS, ELBOW, or FOREARM 41 77%
SHOULDER or SUBSCAPULA 15 28%
NECK 11 20%
10 20 30 40 50
Number of Patients

Fig. 2.—Distribution of pain or paresthesia in 53 patients.

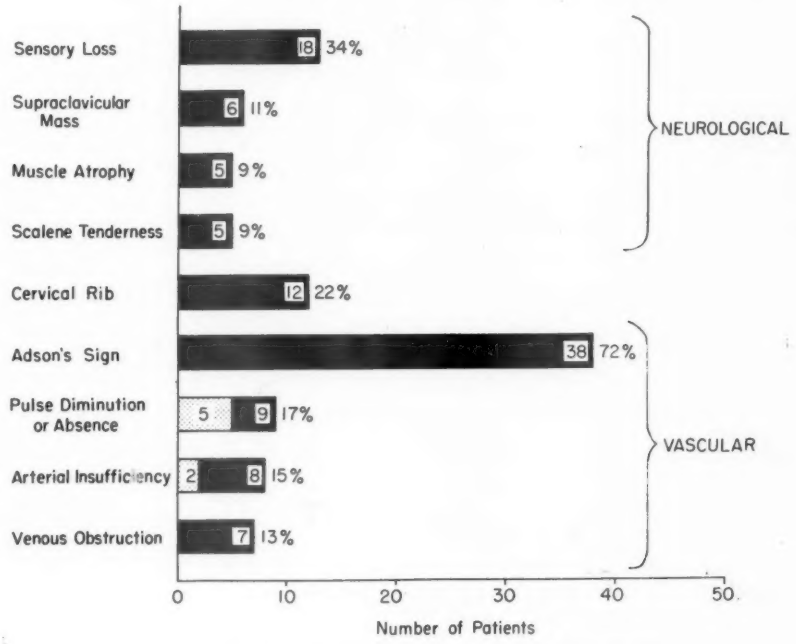


Fig. 3.—Objective findings in 53 patients.

clavicular region in 6 patients while muscle atrophy was found in 5 patients. Muscles of the interosseus group were atropic in 2 patients, while the hypothenar muscles were involved in 3 instances. Tenderness to palpation directly over the scalene muscle was present in 5 patients.

Some objective evidence of vascular compression occurred in 44 patients (83 per cent). Adson's maneuver produced a change in the radial pulse in 38 patients while a persistent diminution in radial pulse was present in 9 patients. The radial pulse was absent in 5 of this group. Evidence of arterial spasm was seen in 8 instances with actual gangrene of the fingers in 2 patients. One of these 2 patients has been previously reported.[2] Both of them support the concept advanced by Leriche[3] that long-standing extrinsic pressure on the wall of the subclavian artery may initiate arteriospasm in the distal artery severe enough to cause occlusion of the radial pulse. Seven patients are included with subclavian vein obstruction with and without radiating pain. The subclavius muscle and tendon were removed in this group and in addition an anterior scalenotomy was required.

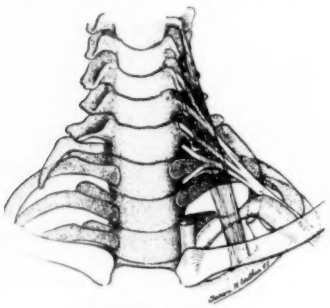


Fig. 4.

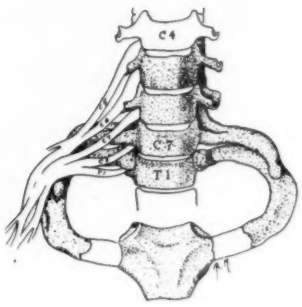


Fig. 5.

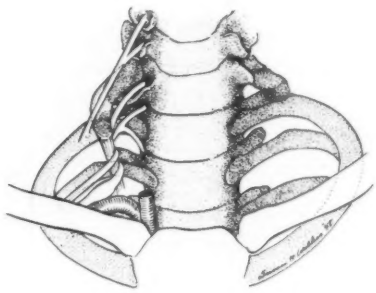


Fig. 6.

Fig. 4.—Cervical rib with compression of brachial plexus and subclavian artery.
Fig. 5.—Long transverse process of seventh cervical vertebra with nerve root compression.
Fig. 6.—Fibrous band extending from cervical rib to first rib.

Operative findings are varied (Table I). Cervical ribs were found in 12 patients (Fig. 4) while an abnormally long transverse process of the seventh cervical vertebra was responsible for the compression in 2 patients (Fig. 5). The scalenus medius was thought to be implicated in only one instance. A fibrous band extended across the subclavian artery in 3 patients (Fig. 6). Abnormal anatomic variation in the scalenus anticus muscle occurred in 3 patients with tendinous incorporation of the subclavian artery.

TABLE I. OPERATIVE FINDINGS IN 53 PATIENTS

	NUMBER OF PATIENTS
1. Cervical rib	12
2. Compression of subclavian artery	13
3. Compression of brachial plexus	11
4. Compression of subclavian vein	7
5. Elongated transverse process (C_7)	2
6. Fibrous band	2
7. Abnormal scalenus anticus	3
8. Abnormal scalenus medius	1
9. Incorporation of nerve or artery in scalenus	2
10. Poststenotic dilatation of subclavian artery	4
11. Thrombosis of subclavian artery, bilateral	1

Marked compression of the subclavian artery by the scalenus anticus muscle was found in 13 patients while the brachial plexis trunks were compressed visibly in 11 patients. The subclavian vein was thought to be compressed by the subclavius muscle, subclavius tendon, or anterior scalene muscle in 7 patients (Fig. 7). Poststenotic dilatation of the subclavian artery was found in 4 patients (Fig. 8). None has progressed to aneurysm or required further treatment. One patient with subclavian artery thrombosis required a bypass arterial graft.

Fig. 7.

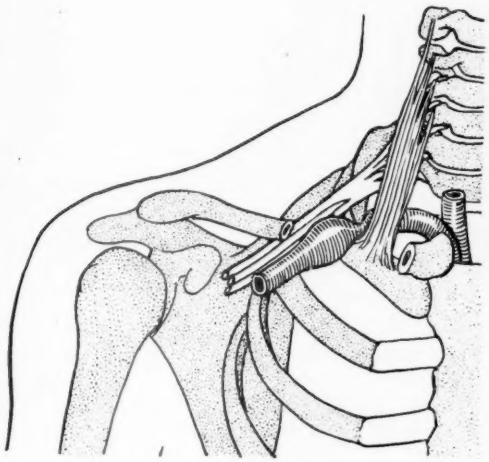


Fig. 8.

Fig. 7.—Venogram demonstrating obstruction of subclavian vein at level of first rib caused by compression of subclavius muscle or tendon and scalenus anticus muscle.
Fig. 8.—Poststenotic dilatation of subclavian artery.

TABLE II. OPERATIVE PROCEDURES IN 53 PATIENTS

	NUMBER OF PATIENTS
1. Anterior scalenotomy (all patients)	53
2. Excision of cervical rib	10
3. Excision of subclavius muscle	7
4. Anterior scalenotomy with preservation of cervical rib	2
5. Cervicodorsal sympathectomy	5
6. Excision of transverse process (C_7)	2
7. Excision of fibrous band	2
8. Anterior and middle scalenotomy	1
9. Resection of first and second ribs	1
10. Arterial graft (bypass)	1

TABLE III. RESULTS IN 53 PATIENTS WITH THORACIC OUTLET COMPRESSION

		PRESENT STATUS			
	PATIENTS FOLLOWED	GOOD	FAIR	POOR	NO FOLLOW-UP
1. Scalenus anticus compression	42	33 (80%)	8 (18%)	1 (2%)	4
A. No cervical rib (Group I)	34	26	7	1	3
B. Cervical rib (Group II)	12	11	0	0	1
2. Subclavian vein compression (Group III)	7	6	1	0	0

The operative procedures are varied also (Table II). Anterior scalenotomy was carried out in all of the 53 patients. Although cervical ribs were present in 12 patients an excision was done in only 10 instances (Table III). Cervicodorsal sympathectomy was performed in 5 cases because of vascular phenomena usually consisting of persistent arterial spasm or occlusion. In 4 of these a cervical rib or bony abnormality was present, but the fifth patient presented some unusual findings in the absence of rib anomaly.

CASE 1.—VUH 241364, a 49-year-old white woman, had onset of coldness and cramping pain in her left hand 3 weeks before admission to Vanderbilt University Hospital. Some numbness of the third, fourth, and fifth fingers was noted at times. There was no history of trauma. No pulses were felt distal to the axillary artery.

During the induction of cyclopropane anesthesia the left radial pulse was felt and the blood pressure just above the left elbow approximated the pressure on the right. A thick tendinous anterior scalene muscle was divided, relieving definite compression on the subclavian artery and lower part of the brachial plexus. The inferior cervical ganglion and sympathetic chain were exposed and the chain divided below T_3 and the proximal end sutured in the wound after division of all rami.

Immediately after operation the left brachial pulse was palpable but the radial pulse was not felt until a check-up examination 7 weeks later. Blood pressure in the left arm was 140/70 compared to 110/70 on the right, while the oscillometric readings were 6 on the right arm and 2 on the left.

This patient subsequently developed extreme arteriospasm of the lower extremities after exposure to cold. No pulses distal to the femorals could be felt. The terminal aorta and common iliac arteries were normal when palpated during the course of lumbar sympathectomy.

The subclavius muscle and tendon were removed in addition to anterior scalenotomy in the 7 patients with subclavian vein obstruction. The long seventh cervical transverse process was removed on two occasions. The scalenus medius muscle was divided in one patient only. The first rib and a portion of the second rib were resected in an individual with a high first rib which was producing compression. One patient was found to have bilateral subclavian artery thrombosis commencing behind the anterior scalene muscle and an arterial bypass graft was inserted in addition to anterior scalenotomy.

CASE 2.—VUH 291704 (courtesy of Dr. H. William Scott), a 29-year-old white housewife, was admitted to Vanderbilt University Hospital with a chief complaint of weakness of her arms for the past year. Elevation of her arms for even a minute caused weakness and blanching of her hands. There was a history of attacks of syncope since puberty. Examination revealed absence of pulses in both upper extremities and decreased in the lower extremities. Retrograde aortograms showed a segmental block in both subclavian arteries distal to the thyrocervical trunk. The carotid arteries were smaller than normal. There were no cervical ribs or bony abnormalities.

At operation the right brachial artery was found to be about the size of the ulnar nerve but contained bright red blood. The subclavian artery was found to be occluded behind the anterior scalene muscle which was divided. The space between the high first rib and the clavicle was minimal and the tip of the operator's finger was pinched when the arm was abducted. The occluded subclavian artery was incised and a well-organized thrombus found. Proximal thromboendarterectomy for about 5 cm. was done with a vigorous gush of blood as the clot was removed. Since the clot could not be removed distally, a woven Teflon graft was sutured to the opening in the subclavian and then to the brachial artery, end-to-side. Postoperatively there was a good brachial pulse but neither radial nor ulnar arteries were palpable. Anticoagulants were given after operation.

There seems little doubt about the compression of the subclavian artery between the first rib and clavicle. Following anterior scalenotomy this space was greatly increased. It is of interest that removal of the proximal thrombus resulted in a stronger right carotid pulsation and no syncopal attacks have occurred.

While advanced arterial changes are more likely to be associated with bony abnormalities, soft tissue compression of the artery may certainly result in local and even distal thrombosis.

RESULTS

The present status of 49 of the 53 patients is known (Table VI). Of the 42 patients presumed to have artery or nerve compression (Groups I and II) there are 33 (80 per cent) who have good results. Eight patients (18 per cent) continue to have moderate symptoms but this is not disabling in 7 patients. All but this remaining one are improved but by varying degrees. One patient (2 per cent) continues to have similar pain of equal severity as before operation which cannot be attributed to postoperative or traumatic neuralgia. This patient did not have a cervical rib but had only equivocal objective signs of vascular or nerve root compression. Her subjective symptoms were much more prominent than any objective finding.

Of the 11 patients followed in Group II with a cervical rib there was satisfactory relief of symptoms in each case. Six of these 7 patients with subclavian vein compression received relief from symptoms following operation. The one patient in this group (Group III) who has a "fair" result only had "effort thrombosis" of the subclavian vein, and as one of the sequelae he has recurrent edema of the hand and forearm.

It is of interest that the 2 patients who had preservation of the cervical rib had good results. In one patient the rib was quite short and did not make contact with the artery or nerve trunks. The other patient had a long cervical rib but division of the anterior scalene muscle evidently allowed the first rib to descend enough to relieve compression. We feel that a cervical rib presents a hazard for the present or future that justifies its removal in all symptomatic cases.

While the average duration of symptoms was 27.5 months for the 53 patients, the average was only 4.2 months for those 8 patients who have persistence of symptoms. This suggests that careful preoperative evaluation and repeated examination may eliminate some of the undesirable candidates for operation.

COMMENT

The sites of potential neurovascular compression of the upper extremity include the interscalene triangle, the costoclavicular space with its ligament and subclavius muscle, the axilla containing the pectoralis minor muscle near its coracoid insertion and the "sling" formed around the axillary artery by the two heads of the median nerve.[4]

Factors contributing to symptoms from these compression sites may be mechanical motion or angulation, hypertrophy or atrophy of muscles, trauma,

or simply developmental in character such as a cervical rib, bony protuberance, or fibrous band. Unfortunately the symptoms produced by compression at these various sites are neither consistent nor diagnostic of the underlying anatomic abnormality. Pain due to vascular compression frequently cannot be distinguished from pain caused by nerve root compression. Conversely objective vascular phenomena may be produced by nerve root stimulation. In essence, then, one must be prepared to explore each area of potential compression if the initial operative findings do not account reasonably for the patient's symptoms and signs. Thorough exploration of the artery, vein, and nerve trunks in this area is mandatory. Scalenotomy, excision of a cervical rib or bony prominence, division of fibrous bands and ligaments, or occasionally removal of the subclavius muscle and tendon may be indicated in any of these combinations.

The application of cervicodorsal sympathectomy in this group of patients should be mentioned. We have used sympathectomy as an adjunct when evidence of vasospasm, thrombosis, or suspected arterial insufficiency is present. A transaxillary approach has proved very satisfactory for cervicodorsal sympathectomy for other conditions.

There were 5 patients who had a cervicodorsal sympathectomy in addition to scalenotomy. Three of these had a sympathectomy at the time of scalenotomy and through the same incision. The other two had a Smithwick-type sympathectomy through a posterior approach several months after division of both anterior scalene and subclavius muscles and tendons. These two had marked hyperhidrosis in addition to previous subclavian vein compression. Only one of these 5 patients had cervical ribs but all had marked vascular changes. All were definitely benefited by sympathectomy and there were no complications as a result of the procedure.

Lord and Rosati[4] emphasized the dangers of sympathectomy through a supraclavicular (Telford) approach. They reported a case of gangrene of the hand due to "reflex arteriospasm following an upper thoracic sympathectomy carried out by the supraclavicular approach" and warn against its use in a patient with vascular impairment. They prefer the anterior transthoracic approach described by Palumbo.[7]

Falconer and Weddell[1] have described costoclavicular compression and have separated it sharply from the scalenus anticus syndrome. The backward and downward thrust of the shoulder exerts pressure on the subclavian artery and vein from the closure of the angle between the clavicle and first rib. Lord[4] has reviewed this area extensively since he feels also that it is the major source of difficulty in patients with thoracic outlet compression.

In our experience venous occlusion is more likely to be found with costoclavicular compression while artery and nerve compression are more likely to occur at the scalene triangle.

While many patients with subjective evidence of thoracic outlet compression are examined each year by two of us (J. A. K. and D. H. R), we have come to feel that only in patients with objective signs are the operative results likely to be

satisfactory. Only 3 of the 53 patients in this series had subjective evidence alone and two of them have been placed in the "fair" and "poor" result category.

CONCLUSIONS

1. Fifty-three patients with thoracic outlet compression were treated by anterior scalenotomy with or without another adjunctive procedure.

2. Of the 42 patients who had evidence of nerve or artery compression, 80 per cent received relief of symptoms.

3. Of the 7 patients who had evidence of subclavian vein compression, 6 received relief of symptoms.

4. In the selection of patients for operative treatment it is desirable to have objective evidence of neurovascular compression rather than subjective symptoms alone.

5. Cervical ribs were present in 12 patients (23 per cent). It is advisable to remove the cervical rib when symptoms attributable to neurovascular compression are present.

6. Cervicodorsal sympathectomy with scalenotomy may be indicated in certain instances of arterial occlusion, thrombosis, or spasm.

7. Careful preoperative evaluation with objective signs may reduce the number of unsatisfactory results following operation.

8. Thorough exploration of the entire area is necessary to assure acceptable results.

REFERENCES

1. Falconer, M. A., and Weddell, G.: Costoclavicular Compression of the Subclavian Artery and Vein Relation to Scalenus Anticus Syndrome, Lancet **2**: 539, 1943.
2. Kirtley, J. A., Jr.: Some Results of Arterial Spasm, South. Surgeon, **16**: 645, 1950.
3. Leriche, R.: Surgery of Pain, Baltimore, 1939, The Williams & Wilkins Co., p. 238.
4. Lord, J. W., Jr., and Rosati, L. M.: Neurovascular Compression Syndromes of the Upper Extremity, Clinical Symposia, Ciba, **10**: 1958.
5. McCleery, R. S., Kesterson, J. E., Kirtley, J. A., Jr., and Love, R. B.: Subclavius and Anterior Scalene Muscle Compression as a Cause of Intermittent Obstruction of the Subclavian Vein, Ann. Surg. **133**: 588, 1951.
6. Ochsner, A., Gage, I. M., and De Bakey, M. E.: Scalenus Anticus (Naffziger) Syndrome, Am. J. Surg. **28**: 669, 1935.
7. Palumbo, L. T.: Anterior Transthoracic Approach for Upper Thoracic Sympathectomy, A. M. A. Arch. Surg. **72**: 659, 1956.

Discussion

DR. ALTON OCHSNER (New Orleans, La.).—I agree with Dr. Riddell that in most instances all one need do is a resection of the scalenus anticus, and I would advise doing a resection rather than a simple myotomy.

In our original report, which we made some time ago, we had not had any recurrences at that time, but in following these cases later we found some recurrences. In each instance it was due to the fact that the defect between the divided scalenus anticus muscle and the first rib had been bridged by scar. It is probably very much the same thing that happens when one performs a division of the sternocleidomastoid muscle for torticollis. The orthopedists have learned that simple division of the sternocleidomastoid is not sufficient, but that they must excise the muscle rather widely. If one is to avoid recurrence, one should resect a considerable portion of the scalenus anticus muscle.

I, too, believe that in some instances it is necessary to resect the cervical rib. In many patients, it is not because the cervical rib slants downward. The symptoms in most

instances are caused by a pinching of the neurovascular bundle between the cervical rib and the attachment of the scalenus anticus on the first rib. If one will simply excise part of the muscle, as Adson originally described, the neurovascular bundle can slide forward and the compression effect is obviated.

DR. GEORGE A. YEAGER (Baltimore, Md.).—Dr. Riddell, in your results that you classified as only fair or poor, have you considered the possibility of a pectoralis minor compression? We have had occasion in the past year or two to re-evaluate some of our patients with poor results from scalenotomy. Resection of the pectoralis minor muscle has resulted in improved results. In all shoulder girdle syndromes this is one factor that should be evaluated.

DR. H. GURTH PRETTY (Montreal, Canada).—I appreciate Dr. Ochsner's remarks on removing a fair amount of the scalenus anticus muscle. I find that in certain cases one is well advised to section and remove about 2 inches of the scalenus medius as well, thus eliminating as much additional scar tissue as possible.

Today when reading reports from a medicolegal aspect, we frequently encounter a condition referred to as a whip-lash injury of the neck. Is it an entity or a coined word to suit our legal associates who wish to impress a jury? Does such a condition exist? I have seen many such cases in consultation. They are protracted, do not readily respond to a Thomas' collar, or physiotherapy and may cause neck and shoulder pain for months. In my opinion, the term "whip-lash injury" should be discarded. The condition is a scalene syndrome secondary to trauma. It responds readily to surgery.

In the mining industry miners are frequently struck by falling timbers or rock at the base of the neck and the shoulder. This injury often produces tears of the scalene muscles and in some cases also comminuted fractures of the clavicle. Relief of symptoms is brought about by resection of the middle third of the clavicle or resection of the scalene muscles and in some cases a combined procedure.

Please bear in mind the possibility of a scalene syndrome when a whip-lash injury is referred to.

DR. VICTOR DEWOLFE (Cleveland, Ohio).—It is very important to remember that there are a large number of patients with the so-called "outlet" syndrome, who have extremely mild symptoms and will not require surgery. These are the individuals who complain of paresthesias with the arm in certain positions, and if pain is present it is mild. If their arm is put in certain positions, the patient will say that the arm "goes to sleep," and when it is brought down to a neutral position, the arm will "wake up" again.

These patients respond extremely well to a well-outlined program of physical therapy, largely posture training and exercises to correct posture and strengthen the elevators of the shoulders.

When many of these patients are told to avoid putting their arm in abnormal positions, they have little difficulty and with this physical therapy program do extremely well.

DR. RIDDELL (closing).—In answer to Dr. Yeager's question about the pectoralis minor compression, we have not divided the pectoralis minor, nor have we felt that it played a part in the particular 8 patients in whom we feel undesirable results are present. Most of these patients, all but one, have actually been improved. Just one of the group is unimproved.

I certainly agree with Dr. deWolfe, and the body of the paper will expound on that somewhat, that a large number of people will be seen with subjective symptoms of neurovascular compression who need never come to surgery for this particular complaint and who can be handled conservatively. While the average duration of symptoms was 27 months for 53 patients, the average was only 4.2 months for the 8 patients who have a persistence of symptoms. This in itself is worth mentioning in that it suggests that a careful preoperative evaluation and repeated examinations may eliminate some of the more undesirable candidates for operation.

THE DEMONSTRATION AND QUANTIFICATION OF REVERSE FLOW IN THE PROXIMAL PORTAL VEIN FOLLOWING SIDE-TO-SIDE PORTACAVAL ANASTOMOSIS

ROBERT T. L. LONG, M.D., CARLOS R. LOMBARDO, M.D., AND ANDREW G. MORROW, M.D., BETHESDA, MD.

(From the Clinic of Surgery, National Heart Institute)

IT has been generally assumed that following the construction of a side-to-side portacaval anastomosis, blood from the portal vein continues to flow to and perfuse the liver. Recent clinical observations, however, have suggesed that flow in the portal vein, cephaled to the anastomosis, may be either bidirectional or reversed. Longmire and associates[7] recovered high concentrations of radioactive serum albumin from this segment of the portal vein after it had been injected into the hepatic artery in patients with side-to-side anastomoses. Child and Donovan,[2] on the basis of pressures recorded in the portal stump after end-to-side portacaval anastomosis, also concluded that retrograde flow occurred in the portal vein after side-to-side anastomosis. In the present experimental study the various components of hepatic blood flow were directly measured in normal dogs after the establishment of side-to-side and end-to-side portacaval communications.

MATERIALS AND METHODS

Healthy, fasting mongrel dogs weighing 14 to 35 Kg. were employed. Anesthesia was induced and maintained with intravenous thiopental. A cuffed endotracheal tube was inserted and positive pressure respiration was maintained with 100 per cent oxygen. The right pleural cavity was entered through the sixth intercostal space. The abdomen was opened through a right thoracoabdominal incision extending from the tenth intercostal space through the right rectus muscle to the midline of the abdomen and the right hemidiaphragm was transected to expose the liver. Heparin (3 mg. per kilogram of body weight) was given intravenously immediately before the cannulations shown diagrammatically in Fig. 1 were carried out.

The portion of the inferior vena cava receiving hepatic venous blood was bypassed by a large plastic cannula inserted into the right atrium and distal inferior vena cava. The gastroduodenal and right gastric arteries were ligated near their origin from the hepatic artery. The hepatic artery was cannulated with two polyvinyl catheters placed proximally and distally and all hepatic artery flow passed through a recording rotameter. Polyethylene cannulae 7 mm. in diameter were inserted into the side of the portal vein just below the entrance of the splenic vein and into the thoracic portion of the inferior vena cava. The bypassed segment of vena cava, receiving hepatic venous drainage, was then isolated between ligatures placed around it above the renal veins and at its junction with the right atrium. Flow from the isolated caval segment and from the portal vein drained into separate graduated cylinders. The pressures in these cannulae were adjusted to an identical level of 0 to 5 mm. Hg by adjustment of screw clamps on them. The physiologic equivalent of a side-to-side portacaval anastomosis was thus established. Resistance to flow through the shunt cannula

Read at the Thirteenth Annual Meeting of the Society for Vascular Surgery, Atlantic City, N. J., June 7, 1959.

was minimal, as evidenced by the fact that when it was open portal vein and venae cavae pressures were equal. When the portal vein was occluded cephalad to the drainage cannula, the preparation was converted to an end-to-side shunt. The cylinders were connected at their bases by a T tube to a one liter reservoir of heparinized venous blood. The flow from the hepatic veins and from the shunt was returned to the jugular vein from the venous reservoir by an occlusive pump which was manually regulated to maintain the reservoir level constant. Mean arterial pressure was monitored throughout the study and maintained at a uniform level by transfusion when necessary. Flows from the hepatic veins and from the portal vein ("shunt flow") were determined from timed collections in the collecting cylinders. The sum of these flows represented not only total hepatic blood flow but also that entering the isolated caval segment from diaphragmatic and lumbar veins.

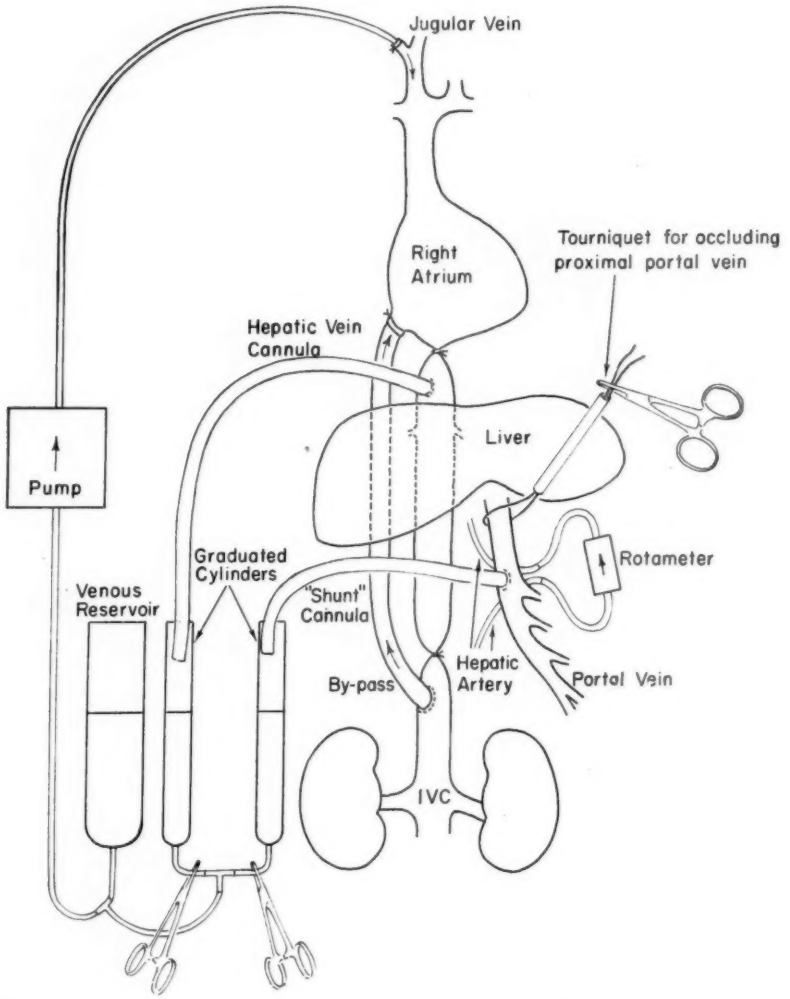


Fig. 1.—Diagrammatic representation of the experimental method employed to permit the establishment of end-to-side and side-to-side portacaval shunts and the measurement of the components of hepatic blood flow. The cannulations and procedure are described in the text.

In the presence of a side-to-side shunt, portal venous flow (from the splanchnic bed to the anastomosis) could be calculated from the equation:

Hepatic arterial flow + portal venous flow = hepatic vein flow + shunt flow.

In order to calculate the true hepatic venous flow more accurately, an estimation was made of that portion of the flow from the caval segment which was due to diaphragmatic and

segmental lumbar venous blood when an end-to-side shunt was present. The magnitude of this extrahepatic venous return was determined by subtracting hepatic arterial flow from the hepatic venous flow. The mean of the values obtained for diaphragmatic and lumbar venous flow was used in the same animal to correct the observed hepatic venous flow when a side-to-side shunt was present. Total liver blood flow and its components were measured during a control period (no shunt), with the portal vein drained and unoccluded (side-to-side shunt) and with the proximal portal vein occluded (end-to-side shunt). In each instance flows were measured over two collection periods and after at least 5 minutes had been allowed for equilibration. At the conclusion of each experiment the liver was excised and weighed.

RESULTS

The various components of hepatic blood flow measured in 8 animals are listed in Tables I through III. The flows are expresesed as cubic centimeters per minute per 100 Gm. of liver. When an end-to-side shunt was established (Table I), flow from the isolated caval segment (observed hepatic venous flow) was always greater than measured hepatic arterial flow. This difference was attributed to blood which entered the caval segment from the diaphragmatic and segmental veins. This extrahepatic flow averaged 5.1 c.c. per minute per 100 Gm. of liver in the group of 8 animals, and this value was used to correct the observed hepatic venous flow in Tables II and III. With this preparation "shunt" flow and portal vein flow were, of course, identical. Table II summarizes the data obtained with a functioning side-to-side shunt in the same animals. Under these circumstances shunt flow was consistently greater and corrected hepatic venous flow was far less than with the end-to-side communication. This difference was attributable to retrograde flow from the liver, supplied by the hepatic artery, since portal flow to the anastomosis remained essentially unchanged. The amount of hepatic arterial blood passing retrograde through the portal vein and the shunt was calculated from the formula:

Retrograde hepatic arterial flow = shunt flow (−) portal venous flow.

This value ranged from 9.5 to 21.6 and averaged 14.1 c.c. per minute per 100 Gm. of liver. Expressing this value as a percentage, an average of 71 per cent of hepatic artery flow passed into the proximal vein and through the shunt.

TABLE I. HEPATIC ARTERIAL, HEPATIC VENOUS, AND PORTAL VENOUS FLOWS IN ANIMALS WITH END-TO-SIDE SHUNTS

DOG NUMBER	NUMBER OF OBSERVATIONS	HEPATIC ARTERIAL FLOW	HEPATIC VEIN FLOW (UNCORRECTED)	EXTRAHEPATIC FLOW*	SHUNT FLOW	TOTAL HEPATIC BLOOD FLOW†
1	6	19.2	22.4	3.2	27.4	46.6
2	4	20.2	22.9	2.7	30.5	50.7
3	3	18.7	24.2	5.5	53.5	72.2
4	1	19.8	24.5	4.7	56.5	76.3
5	5	18.0	21.7	3.7	71.7	89.7
6	1	13.6	17.0	3.4	43.2	56.8
7	2	36.1	44.7	8.6	80.9	117.0
8	1	15.6	24.5	8.9	78.7	94.3
Mean		20.1	25.2	5.1	55.3	75.4

All flows are expressed as cubic centimeters per minute per 100 Gm. of liver.

*Extrahepatic flow has been calculated as the difference between measured hepatic venous flow and hepatic arterial flow.

†Corrected for extrahepatic flow.

With the exception of Animal 2 the amount of hepatic arterial flow entering the shunt was significantly greater than that which passed through the hepatic veins. These data indicate that resistance to flow for hepatic arterial blood is less through the portal vein and shunt than through the sinusoids and the hepatic veins in the normal dog when a side-to-side portacaval communication is present.

TABLE II. FLOW DATA FROM DOGS WITH SIDE-TO-SIDE PORTACAVAL SHUNTS

DOG NUMBER	NUMBER OF OBSERVATIONS	HEPATIC ARTERIAL FLOW	MEASURED HEPATIC VENOUS FLOW	EXTRAHEPATIC FLOW	SHUNT FLOW	PORTAL VENOUS FLOW	CORRECTED HEPATIC VENOUS FLOW*	HEPATIC ARTERIAL FLOW THROUGH SHUNT	TOTAL HEPATIC BLOOD FLOW*
1	6	18.7	7.8	3.2	46.8	32.7	4.6	14.1	51.4
2	6	20.0	13.2	2.7	44.5	35.0	10.5	9.5	55.0
3	5	19.8	10.7	5.5	72.8	58.2	5.2	14.6	78.0
4	3	17.0	5.6	4.7	74.2	58.1	0.9	16.1	75.1
5	5	19.6	9.2	3.7	84.5	70.4	5.5	14.1	90.0
6	1	14.0	7.2	3.4	57.0	46.8	3.8	10.2	60.8
7	3	36.0	23.0	8.6	96.5	74.9	14.4	21.6	110.9
8	1	15.6	12.1	8.9	90.5	78.1	3.2	12.4	93.7
Mean		20.1	11.1	5.1	71.0	56.8	6.0	14.1	76.8

The values are expressed as cubic centimeters per minute per 100 Gm. of liver.
*Corrected for extrahepatic flow.

TABLE III. COMPONENTS OF TOTAL HEPATIC BLOOD FLOW WHEN NO SHUNT WAS PRESENT

DOG NUMBER	NUMBER OF OBSERVATIONS	TOTAL HEPATIC BLOOD FLOW* (C.C./MIN./ 100 GM. OF LIVER)	TOTAL HEPATIC BLOOD FLOW* (C.C./MIN./ KG. OF BODY WEIGHT)	HEPATIC ARTERIAL (PER CENT OF TOTAL)	PORTAL VENOUS (PER CENT OF TOTAL)
1	1	42.5	13.3	34.5	65.5
2	2	48.2	16.8	39.9	60.0
3	1	72.3	14.0	20.0	80.0
4	1	74.5	26.7	19.7	80.3
5	2	71.3	23.1	22.5	77.5
6	1	47.8 X	13.0 X	26.1 X	73.9 X
7	2	104.0	22.1	32.1	67.9
8	1	72.8	17.3	20.5	79.5
Mean		66.7	18.3	26.9	73.1

*Corrected for extrahepatic flow with exception of values marked X.

Table III lists the values obtained for total hepatic blood flow in each animal before a shunt was created. The values are corrected for extrahepatic flow and in general are somewhat lower than those previously reported.[1, 11]

Fig. 2 illustrates in graphic form the average relative contributions to total hepatic blood flow made by the hepatic artery and portal vein for each preparation. The mean values were 27 and 73 per cent for hepatic arterial and portal venous flow, respectively, when no shunt was present. In the presence of an end-to-side shunt, all hepatic arterial flow passed through the hepatic veins and the portal blood passed via the shunt and these values were identical to those obtained when no shunt was present. In the presence of a side-to-side shunt, however, the shunt flow was increased by that portion of the hepatic arterial blood diverted from the hepatic veins into the portal vein. Hepatic venous return, under these conditions, was reduced proportionately.

DISCUSSION

Porctacaval anastomosis is ordinarily performed for the relief of portal hypertension and to decompress the engorged collateral vessels which accompany it. A successful operation usually accomplishes this objective and ordinarily prevents further episodes of gastrointestinal hemorrhage. The type of anastomosis constructed is unimportant if portal decompression is the sole objective; side-to-side and end-to-side portacaval shunts are equally effective in this regard. Recently, however, attention has been called to the value of side-to-side portacaval anastomosis in the relief of ascites accompanying portal cirrhosis.[8, 10, 12] Hepatic vein wedge pressure has been shown to reflect portal venous pressure and the pressure in the liver sinusoid.[6, 9] Sinusoidal hypertension is a constant feature of portal hypertension due to cirrhosis and indicates that the obstruction to portal venous flow is at or beyond the sinusoids. Hepatic sinusoidal hypertension is not relieved by end-to-side portacaval anastomosis indicating that it is maintained, at least in part, by hepatic arterial flow.[5, 9]

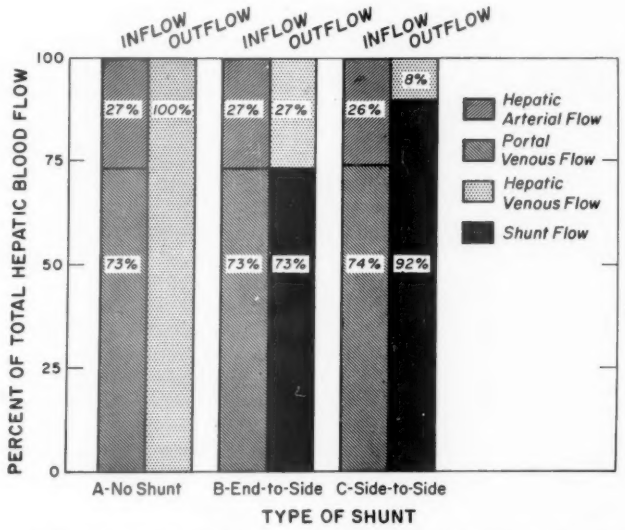


Fig. 2.—Graphic illustration of the components of hepatic blood flow and shunt flow under the experimental conditions studied. Hepatic arterial flow and flow in the portal vein (to the shunt when present) remained constant. The increased shunt flow observed with a side-to-side communication was supplied by hepatic arterial blood which was diverted from the hepatic veins.

Sinusoidal hypertension is probably responsible for the production of liver lymph, an important factor in the etiology of ascites in patients with cirrhosis.[3] The available experimental and clinical evidence thus indicates that sinusoidal decompression can be accomplished by side-to-side anastomosis and that the decompression occurs by retrograde flow of hepatic arterial blood through the portal vein.

Herrick,[4] in 1907, perfused the livers of cadavers and noted that saline flowed from both hepatic and portal veins when the hepatic artery was perfused, even though the portal pressure was maintained at 10 mm. Hg. The amount of perfusate returning via the hepatic veins was greater in the normal livers,

and that returning via the portal was greater in cirrhotic livers. In his studies the level of portal venous pressure was arbitrarily set at 10 mm. Hg during the perfusion and he apparently did not regulate the hepatic venous pressure, which, of course, influences hepatic venous drainage. Under the circumstances of Herrick's experiments with the normal liver, more flow would be expected to occur from the hepatic veins than the portal vein since the resistance to flow through the hepatic veins is less than that through the portal vein. Work in this laboratory, in living animals, indicates that the hepatic arterial and portal venous systems communicate at a pressure equal to the hepatic wedge pressure or sinusoidal pressure. Whether or not this relationship holds true in the dead animal has not been established, and, therefore, perfusion or injection studies similar to those of Herrick's could conceivably introduce changes in pressure dynamics leading to erroneous conclusions.

It was consistently observed that the hepatic wedge pressure in the animals with side-to-side shunts performed by this method or by direct vessel-to-vessel anastomosis was 1.0 to 2.5 mm. Hg greater than that in the decompressed portal vein which was 0.5 to 1.5 mm. Hg greater than vena caval pressure. This indicates that when the portal vein and the hepatic veins drain against an equal pressure the resistance to hepatic arterial flow is less through the portal vein and a shunt (if present) than through the sinusoids and hepatic veins. The shunt receives not only portal venous flow but, in the majority of dogs studied, over 50 per cent of the hepatic arterial blood which passes in a retrograde fashion through the proximal portal vein.

SUMMARY

Side-to-side and end-to-side portacaval communications were established in normal dogs and the various components of hepatic blood flow and shunt flow were measured directly. With a side-to-side shunt, flow through the communication was far larger than when an end-to-side shunt was present. This large difference in shunt flow was supplied by hepatic arterial blood, a major portion of which was diverted directly into the decompressed portal vein. The relationship of these experimental findings to the selection of the type of anastomosis to be performed in patients with portal hypertension and varices or ascites is discussed.

REFERENCES

1. Blalock, A., and Mason, M. D.: Observations on the Blood Flow and Gaseous Metabolism of the Livers of Unanesthetized Dogs, Am. J. Physiol. **117**: 328, 1936.
2. Child, C. G., III, and Donovan, A. J.: Current Problems in Management of Patients With Portal Hypertension, J. A. M. A. **163**: 1219, 1957.
3. Gray, H. K.: Clinical and Experimental Investigations of the Circulation of the Liver, Ann. Roy. Coll. Surg. **8**: 354, 1951.
4. Herrick, E.: An Experimental Study Into the Cause of the Increased Portal Pressure in Portal Cirrhosis, J. Exper. Med. **9**: 93, 1907.
5. Krook, H.: Circulatory Studies in Liver Cirrhosis, Acta med. scandinav. **156**: Suppl. 318: 156, 1956.
6. Levy, C. M., and Gliedman, M. L.: Practical and Research Value of Hepatic Vein Catheterization, New England J. Med. **258**: 696, 1958.
7. Longmire, W. P., Jr., Mulder, D. C., Mahoney, P. S., and Mellinkoff, S. W.: Side-to-Side Portacaval Anastomosis for Portal Hypertension, Ann. Surg. **147**: 881, 1958.

8. McDermott, W. V., Jr.: The Treatment of Cirrhotic Ascites by Combined Hepatic and Portal Decompression, New England J. Med. 259: 897, 1958.
9. Taylor, W. Jape, and Myers, J. D.: Occlusive Hepatic Venous Catheterization in the Study of the Normal Liver, Cirrhosis of the Liver and Noncirrhotic Portal Hypertension, Circulation 13: 368, 1956.
10. Theron, P., and Allen, J. C.: Ascites, a Clinical and Experimental Study, Acta med. scandinav. 152: Suppl. 306: 145, 1955.
11. Waldhausen, J. A., Lombardo, C. R., McFarland, J. A., Cornell, W. P., and Morrow, A. G.: Studies of Hepatic Blood Flow and Oxygen Consumption During Total Cardiopulmonary Bypass, SURGERY 46: 1118, 1959.
12. Welch, C. S., Altarian, E., and Welch, H. F.: The Treatment of Ascites by Side-to-Side Portacaval Shunt, Bull. New York Acad. Med. 34: 259, 1958.

DISCUSSION

DR. DAVID M. HUME (Richmond, Va.).—We have been measuring blood flow and pressure in the portal and hepatic venous systems in some of the portacaval shunts we have done lately, and have found that in 3 out of 28 patients there actually has been a retrograde flow in the portal vein even before the shunt was done. In other words, the portal vein had become one of the important outflow tracts of the liver.

One of the questions that is still unanswered is whether this retrograde flow, or the preservation of retrograde flow after a shunt, is a good thing for the patient or a bad thing. One might possibly assume from this paper that retrograde flow was detrimental since it diverts arterial blood from its task of nourishing the cells of the liver. However, if the patient has developed a retrograde flow of blood out of the liver through the portal vein before he is operated upon, as a consequence of contraction of the hepatic vein system, and the portal vein is then tied off as would be done with an end-to-side shunt, this would certainly create hepatic hypertension, which is probably bad for the patient. A side-to-side shunt under these circumstances would permit the liver to become decompressed by allowing a great deal of the blood to flow out of the liver through the shunt.

Drs. Muller and Warren of the University of Virginia have done some studies which are pertinent to the discussion and which indicate that after a side-to-side shunt the hepatic cells remove oxygen from the blood going out of the portal vein. In other words, the blood that flows out of the portal vein does nourish the hepatic cells to some extent, and is not altogether lost to the cells of the liver.

CONTINUING STUDIES OF REPLACEMENT OF SEGMENTS OF THE VENOUS SYSTEM

ROBERT BOWER, M.D., VALERIO FEDERICCI, M.D., AND JOHN M. HOWARD, M.D.,
PHILADELPHIA, PA.

(From the Department of Surgery, Hahnemann Medical College and Hospital)

DEVELOPMENT of a satisfactory graft for venous replacement is an objective long recognized by vascular surgeons. Experimental attempts using homografts and various plastic prostheses have been reported and have met with only slight success.[2-5] It has been our purpose further to elucidate the factors which cause a homograft or prosthesis to fail as a vein, where a vein autograft in the same situation would succeed.

Since for the present we cannot obviate the host tissue reaction to a foreign graft, we have sought to determine how we can control the specific aspects of this reaction which result in graft failure.

MATERIALS AND METHODS

Adult female mongrel dogs, weighing 15 to 30 Kg., were used for implantation of the grafts. Veterinary Nembutal 0.4 c.c. per kilogram was administered intravenously to induce surgical anesthesia. An endotracheal tube was inserted immediately after induction of anesthesia and was left in place for the duration of the procedure. If at any time the animal showed signs of respiratory difficulty, a mechanical respirator was attached to the endotracheal tube and assisted respiration was continued for as long as was necessary. Tracheal aspiration was performed periodically during the procedure and before the removal of the tube.

The inferior vena cava was exposed and dissected out from the junction of the iliac veins below to the renal veins above, a distance of 8 to 10 cm. Two or three tributaries, usually one anteriorly and two posteriorly, were ligated and divided. Pott's coarctation clamps were applied above and below and 5 mg. of heparin was injected below the lower clamp and above the upper clamp. A segment of vein was excised leaving at least a 1 cm. cuff of vein extending beyond the clamp at both ends. Failure to leave a long enough cuff or shortening of the cuff by the clamps slipping toward the cut end rendered the anastomoses unnecessarily difficult in some instances. A homograft or prothesis approximately 25 per cent longer than the excised segment was then inserted using a quadrangulation technique with or without beveling of the ends.

At the conclusion of the lower anastomosis the upper clamp was removed, followed in a few seconds by removal of the lower clamp. Blood loss from the suture lines and through the fabric of the prostheses was minimal.

The animals received no special care postoperatively pertaining to the grafting procedure. Venograms were performed at varying intervals after insertion of the grafts by injecting 15 to 20 c.c. of 90 per cent Renografin into a vein in one hindleg.

At the time of autopsy the graft area was inspected for evidence of hemorrhage, infection, and excessive fibrosis. The grafts were removed intact with adjacent segments of host vein and examined externally and intraluminally.

Read at the Thirteenth Annual Meeting of the Society for Vascular Surgery, Atlantic City, N. J., June 7, 1959.

RESULTS

Forty-six animals were divided into four groups as follows: Group I cross-clamp controls, Group II autografts, Group III Teflon and nylon prostheses, and Group IV homografts.

Group I. Cross-Clamp Controls.—Eleven animals had an operation performed similar in every way to a graft procedure except that after application of the coarctation clamps to the vena cava one hour was allowed to elapse and the clamps were removed. No thromboses occurred in this group.

Group II. Autografts.—Eleven animals had a 3 to 5 cm. segment of inferior vena cava excised and replaced as an autograft. One graft was thrombosed at 9 days. The remaining 10 autografts have remained patent and functionally adequate for periods up to 8 months (Table I). Four of these animals died or were sacrificed 6 to 16 weeks after grafting. Examination of the graft sites revealed minimal perivenous fibrosis. Examination of the removed grafts showed smooth suture lines with slight reductions in diameter at the anastomoses. No intraluminal thromboses were found in the grafts or at the suture lines.

TABLE I. RESULTS WITH AUTOGRAFTS

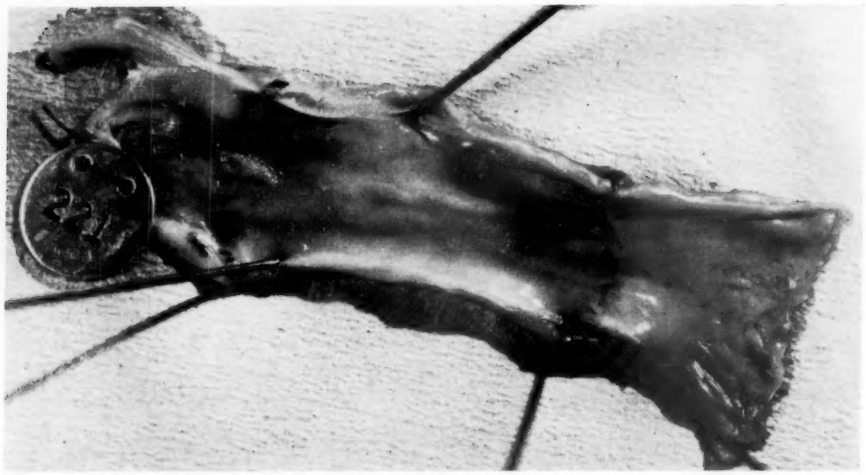
GRAFT NUMBER	VENOGRAM DAY	RESULT OF VENOGRAM	REMARKS
A–36	2	Patent	Patent at 6 mo.
		Patent	
A–38	16	Patent	Autopsy at 4 mo.,
	23	Patent	graft patent
A–28	14	Patent	Living
	21	Patent	
A–41	9	Thrombosis	
A–44	7	Patent	Living
	14	Patent	
A–42	14	Patent	Living
A–39	2	Patent	Living
A–32	21	Patent	Sacrificed 4 mo., graft patent
A–33			Autopsy 40 days, graft patent
A–40	77	Patent	Autopsy 11 wk., graft patent
A–19	48	Patent	Living

Group III. Prostheses.—Eight animals had segments of the inferior vena cava replaced with crimped Teflon. One animal was sacrificed on the fifth postoperative day and was found to have a patent functioning graft. The remaining 7 grafts all thrombosed in the first few days after insertion (Table II). At autopsy the grafts were found to be occluded with thromboses with slight-to-moderate stenosis at the suture lines.

Six animals had caval grafts of seamless nylon. Two of these grafts remained patent for extended periods and showed good functional capacity. One animal was sacrificed at 125 days; the graft had a smooth lumen without

evidence of stenosis at the suture lines.* The second animal is living with a patent graft after 8 months. The other 4 grafts completely thrombosed in the postoperative period. At autopsy longitudinal wrinkling of the grafts was found with much surrounding fibrosis. The suture lines were markedly constricted and some intraluminal thrombus was present.

A.



B.

Fig. 1.—*A*, Venogram of nylon prosthesis 3 months after insertion. *B*, Nylon graft removed from same animal after 4 months. Note smooth intimal surface and longitudinal wrinkling.

Group IV. Homografts.—Venous homografts from 3 to 6 cm. in length were inserted in 10 animals. One graft remained patent and functionally adequate for 5 months until death occurred following venography (Fig. 2, *A*).

*A venogram taken 3 months postoperatively and the graft removed at autopsy 4 months postoperatively are shown in Fig. 1.

At autopsy the appearance of the homograft was quite similar to an autograft with minimal perivascular fibrosis and slight narrowing of the suture lines (Fig. 2, *B*). A second homograft remained patent until the dog was sacrificed

TABLE II. RESULTS WITH PROSTHESES

GRAFT NUMBER	TYPE OF PROSTHESES	VENOGRAM DAY	RESULT VENOGRAM	DAYS PATENT (ESTIMATE)
A–76	Crimped Teflon	2	Thrombosis	1
A–64	Crimped Teflon	7	Thrombosis	4
A–65	Crimped Teflon	2	Thrombosis	1
A–99	Crimped Teflon	Sacrificed fifth day, graft patent		
A–61	Crimped Teflon	7	Thrombosis	4
102	Crimped Teflon	9	Thrombosis	5
114	Crimped Teflon	2	Thrombosis	1
107	Crimped Teflon	7	Thrombosis	4
A–50	Seamless Nylon	34 97	Patent Patent	Patent at 8 months
A–26	Seamless Nylon	86 121	Patent Patent	Sacrificed 125 days, patent
A–37	Seamless Nylon	7	Patent	Died 20 days, thrombosis
A–35	Seamless Nylon	9	Patent, thrombosis	27
704	Seamless Nylon	2 16	Patent, thrombosis	9
A–29	Seamless Nylon	7	Thrombosis	4

TABLE III. RESULTS WITH HOMOGRAFTS

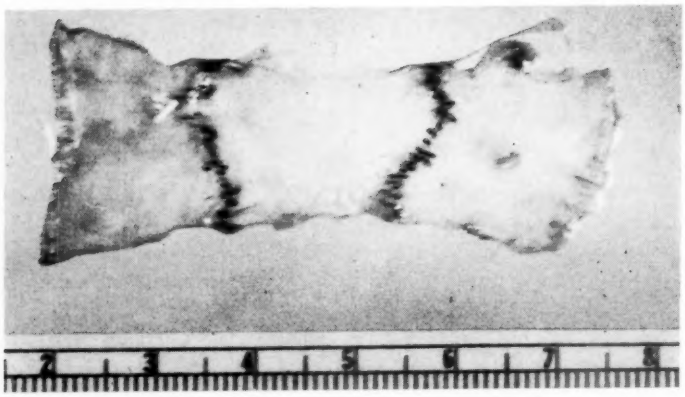
GRAFT NUMBER	VENOGRAM DAY	RESULT OF VENOGRAM	DAYS PATENT (ESTIMATE)
A–34	9 29	Open, thrombosis	19
A–90	9	Thrombosis	5
A–86	7	Thrombosis	4
A–77	9 23	Open, thrombosis	16
A–72	2 16	Open, thrombosis	9
A–81	9	Thrombosis	5
104	7	Thrombosis	4
120	28	Thrombosis	14
1237	4 mo.	Open	Autopsy at 4 mo., patent
A–74	7 30	Open, stenotic	Autopsy at 3 mo., patent

TABLE IV

TYPE OF GRAFT	NUMBER OF GRAFT	NUMBER OPEN	PER CENT OPEN
Cross-clamp control	11	11	100
Vein autograft	11	10	90
Vein homograft	10	2	20
Prostheses	14	3	22

at 2 months but marked constriction of the suture lines had produced functional obstruction. The remaining 8 grafts became completely occluded in the early postoperative period (Table III). Two of the 8 showed patency on venography as long as 9 days postoperatively, but stenosis was already evident and complete occlusion followed shortly. The outstanding findings at autopsy in this group were marked constriction of both suture lines and dense perivascular fibrosis.

A.



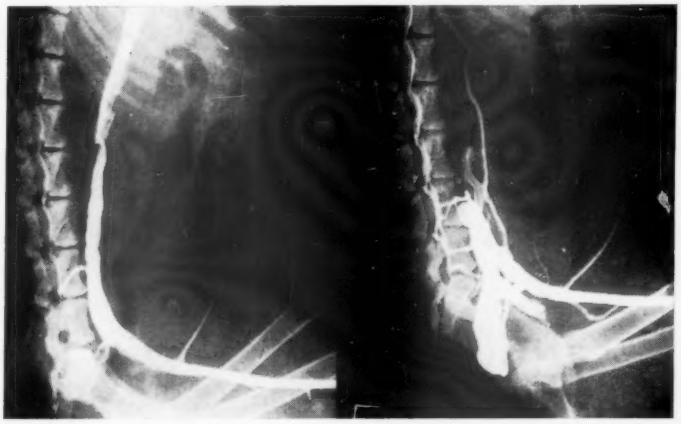
B.

Fig. 2.—*A*, Serial venograms of inferior vena caval homograft 1 week, 1 month, and 2 months after insertion. *B*, Homograft shown in venograms when removed at autopsy after 5 months.

DISCUSSION

A study of our results revealed that occlusion of both prostheses and homografts occurred in the first few days following insertion. It is estimated that the mean duration of patency of crimped Teflon grafts was 3 days, and for homografts about 5 days. Autopsy studies revealed two factors which together were responsible for these occlusions: first, stenosis of the anatomoses and, second, intraluminal thrombosis. Significant stenosis at the suture lines was noted in all grafts which became occluded later than 4 or 5 days after insertion. Earlier than 4 days intraluminal thrombosis was the major finding.

Fig. 3, *B* shows 2 venous homografts which became occluded within a week of insertion. It was observed that the upper (cephalad) anastomosis consistently displayed a greater degree of stenosis than the lower anastomosis, a finding also confirmed by venograms taken before complete occlusion (Fig. 3, *A*).



A.

B.

Fig. 3.—*A*, Venograms of homograft 2 days (*left*) and 2 weeks (*right*) after insertion. Note stenosis at upper anastomosis already apparent by second day. *B*, Showing 2 thrombosed homografts removed at autopsy 3 weeks after insertion. Note marked stenosis of both grafts at the upper anastomosis (indicated by numbered tag.)

We attribute this pattern of stenosis to decreased intraluminal pressure at the cephalad end of the graft produced initially by relative stenosis at the caudad anastomosis and rendered progressively lower by deposition of thrombus there. This is consistent with the observations of Bryant, Lazenby, and Howard,[1] who noted that increasing the intraluminal pressure of a vein graft to

about 50 cm. H_2O by creating an arteriovenous fistula resulted in a higher incidence of patency.

Our studies indicate that both early intravascular thrombosis and delayed stenotic occlusion must be prevented if consistent success is to be realized in venous grafts of approximately 1 cm. in diameter. Adequate anticoagulation deserves a thorough trial in obviating the former. We are currently evaluating the use of rigid anastomotic stents in the form of stainless steel rings for the prevention of delayed stenosis.

SUMMARY

Further experimental observations on the insertion of venous grafts of approximately 1 cm. in diameter are reported. Our control studies confirm the superiority of venous autografts to any known substitute. Intraluminal thrombosis and delayed stenosis at the suture lines were responsible for graft failure when either homografts or plastic protheses were inserted. Means of eliminating these complications have been suggested and are currently under investigation. Sufficient success has been realized with both homografts and certain types of prostheses to warrant optimism about the eventual development of a satisfactory venous substitute.

REFERENCES

1. Bryant, Milton F., Lazenby, W. D., and Howard, J. M.: Experimental Replacement of Short Segments of Veins, A. M. A. Arch. Surg. **76**: 289-293, 1958.
2. Daniel, W. W.: Bridging Defects in Canine Portal and Superior Mesenteric Veins With Plastic Tubes and Vascular Grafts: A Preliminary Report, Cancer **5**: 1041-1048, 1952.
3. Deterling, R. A., Jr., and Bhonslay, S. B.: Use of Vessel Grafts and Plastic Prostheses for Relief of Superior Vena Caval Obstruction, SURGERY **38**: 1008-1026, 1955.
4. Egdahl, R. H., Hume, D. M., and Schlang, H. A.: Plastic Venous Prostheses, S. Forum **5**: 235-241, 1954.
5. Holman, C. W.: Treatment of Superior Vena Caval Occlusion by Arterial Graft: Preliminary Report, J. A. M. A. **155**: 1403-1405, 1954.

DISCUSSION

DR. WILLIAM G. ANLYAN (Durham, N. C.).—Dr. Schauble and I inserted 83 autogenous, homologous, and nylon grafts in the jugular and femoral veins (with and without vena caval ligation) of mongrel dogs. All of the Edwards-Tapp nylon grafts were occluded with organizing thrombi. Forty-two per cent of the fresh-frozen homografts were patent at the 4-month follow-up, but all were occluded after 8 months and found to be embedded in dense fibrous tissue. Up to 80 per cent of our autografts remained completely patent. Isogravitational or antigravitational flow and stasis did not appear to influence the success rate of the autografts.

DR. WILLIAM H. SEWELL, JR. (Atlanta, Ga.).—We've been interested in the problem of replacement of the inferior vena cava because of the Baffes procedure for transposition of the great vessels. As you know, this is classically done by using a homograft to lengthen the inferior vena cava and to deliver its venous blood into the left artrium.

We decided to test Teflon for this purpose and have inserted grafts in a series of 10 dogs. The prosthesis used was of the knitted type and of high porosity. It was sutured to the inferior vena cava and to the atrium by end-to-side anastomoses, just as is done in the Baffes procedure. The only difference was that in the dogs we had to connect the graft to the right atrium rather than the left so that the animals would not die of cyanosis. The Teflon graft was one-half inch in diameter.

As of the time that I left Atlanta all of the dogs were doing very well indeed. They have been followed for a maximum of about 6 months and venograms have been done on some of them from 2 to 4 months postoperatively. The lumina were all very well maintained. None of the dogs developed ascites or other evidence of trouble.

We have not tried using this Teflon prothesis in any human patients but if the dogs continue to do well I believe this might be reasonably attempted.

DR. THOMAS C. MOORE (Muncie, Ind.).—We seem to have had a bit more luck in Indianapolis in keeping our venous grafts open. Our grafts have been in the superior vena cava. It appears that superior vena caval grafts have a greater tendency to remain open than grafts used to replace the inferior vena cava or other segments of the venous system.

We recently have employed fresh homografts of superior vena cava for superior vena cava replacements in 24 dogs. Thirteen of the 24 grafts have remained patent during the period of observation. We have found, in these animals, that the grafts which became occluded did so relatively early after operation. In 8 of the animals repeat cavograms have been carried out. None of these 8 animals in which graft patency was demonstrated at the time of the first cavogram showed occlusion on subsequent cavograms.

We also have been interested in the use of Teflon vascular protheses for superior vena caval replacement, and have carried out 40 of these replacements. The grafts have remained patent in 25 animals (63 per cent). Several sizes of prostheses have been employed and there appears to be a significantly greater degree of success when the larger prostheses are used.

DR. PETER.—We have also been interested in replacing superior vena cava with Teflon fabric, and I should like to present three slides to illustrate our results.

(Slide.) We have replaced a segment of the superior vena cava in 30 dogs. In 7 of them, we put a graft between the superior vena cava and the right atrium. We found after 10 months 17 patent grafts in the superior vena cava series, with 7 partially obstructed and 6 completely obstructed throughout. In the superior vena cava and the right atrium we found only 3 patent grafts after 10 months' follow-up, one partially occluded, and 3 completely occluded.

(Slide.) This is a venogram of a dog which had a graft on the superior vena cava, and after 10 months we can see that the graft is fully patent.

There is some narrowing at both ends of the graft. There is no interruption of collateral circulation on this side as we see when grafts become obstructed.

(Slide.) This is a 13 mm. graft also 10 months old, which shows the inner lining smooth and regular, and continuity of the vessel itself on the right side. It shows also on the other side that the suture line is somewhat narrowed. This type of result has been classified as partially obstructed. You can also see there is some fibrotic reaction along the graft itself.

DR. ANDREW DALE (Nashville, Tenn.).—This investigation of Drs. Howard and Bower brings to mind the interesting difference between thrombosis as it occurs in the arterial and in the venous system.

When thrombosis occurs in the arterial system it is to all intents and purposes final. Blood is carried around the blocked area by collateral circulation. When thrombosis occurs in the venous system, on the other hand, there is very often recanalization.

In a recent and continuing series of approximately 20 dogs with autogenous venous grafts transplanted to the femoral veins and to the inferior vena cava, there has been a significant percentage of initial thrombosis followed by recanalization as determined by phlebograms. All of us who are interested in this field should be aware that an early phlebogram showing such thrombosis may later change and show recanalization and patency if the animal is preserved and followed. This has also been true in the independent series of Dr. James DeWeese which is in press at the moment.

EVALUATION OF THROMBECTOMY IN THE MANAGEMENT OF ILIOFEMORAL VENOUS THROMBOSIS

JAMES A. DEWEESE, M.D., THEODORE I. JONES, M.D., JOHN LYON, M.D., AND W. ANDREW DALE, M.D.,* ROCHESTER, N. Y.

(From the Department of Surgery, University of Rochester School of Medicine and Dentistry)

THE obstruction of the iliac and common femoral veins by thrombosis and the resulting edema of the entire lower extremity are usually painful and may be accompanied by vasospasm or gangrene. Early morbidity results from the painful swelling, particularly with ambulation, and the development of gangrene in a few patients. Mortality may result from pulmonary embolization, massive fluid sequestration, or from complications of the gangrene. Late morbidity may result from chronic edema and pain, recurrent phlebitis, and ulceration.

The aim of an ideal method of treatment for these patients should be to prevent morbidity as well as mortality. Therefore, the ideal method would not only include removing the thrombi from the veins, but would also include restoring normal venous circulation by avoiding venous ligations. With this aim in mind Läwen,[22] in 1938, performed femoral venotomy with thrombectomy and subsequent closure of the venotomy incision to attempt complete restoration of normal venous circulation. Fontaine[16] and Mahorner[25, 26] subsequently reported more extensive experiences with the same procedure and in addition advocated the use of anticoagulants to prevent rethrombosis. Their results indicated that early and late morbidity was decreased and that the mortality was low.

Despite the good results reported from the procedure there is not widely reported acceptance of it in this country. Therefore, the method was evaluated in this clinic and compared with nonoperative methods of management. Thrombectomy was a valuable adjunct in the management of these patients, particularly during the early phases of the disease. The ultimate result was most dependent upon the extent of the disease and the completeness of the thrombectomy. Phlebography proved valuable as a means of evaluating the extent of the thrombosis and the result of the thrombectomy.

METHODS

Clinical Analysis.—Acute iliofemoral venous thrombosis was observed in 27 extremities of 23 patients treated at Strong Memorial Hospital during the period from April, 1955, to April, 1959, as based on the discharge diagnoses in the medical records. The diagnosis was made on the basis of massive edema below the level of the inguinal ligament in association

Read at the Thirteenth Annual Meeting of the Society for Vascular Surgery, Atlantic City, N. J., June 7, 1959.

*Present address: 2118 West End Avenue, Nashville 5, Tenn.

with other signs of deep venous thrombosis. (Thrombosis of the iliac and common femoral veins was further substantiated by either phlebograms, operations, or autopsies in 24 of the 27 extremities.)

The ages of the patients ranged from 20 to 78 years. Thrombosis occurred postoperatively in 8 extremities, and during periods of bed rest for treatment of injuries or other illnesses in 10 other extremities. The process occurred spontaneously in 6 extremities of otherwise healthy individuals. Spontaneous thrombosis also occurred in a pregnant woman near term, and in 2 patients who had had contralateral massive thrombosis several years before. The process involved the left extremity in 15 patients, the right in 4, and occurred bilaterally in the 4 other patients. Treatment was instituted the first day of the disease in 17 instances, within the first 12 days on 8 others, and the twenty-first and twenty-eighth day in the remaining 2. Four of the patients showed evidence of pulmonary embolization prior to the onset of therapy.

Thrombectomy, elevation, and anticoagulation was carried out on 13 extremities of 11 patients. Nonoperative treatment consisting of bed rest, usually anticoagulation, and occasionally sympathetic blocks was instituted in 14 extremities of 12 patients. The reasons for selecting nonoperative management varied. Eight of the extremities were treated prior to the time that thrombectomy was being considered. In 2 other instances the patient's physician preferred not to consider thrombectomy. Nonoperative management was selected in 2 patients (G. G. and L. P.) because of terminal carcinoma, and in one patient who was pregnant and near term. Although the two groups are not strictly comparable in all respects, they are similar and serve to illustrate the problems and results of nonoperative versus operative management.

Origin of Thrombosis.—An analysis of the probable site of thrombosis in patients was made on the basis of history, physical findings, ascending long-film phlebography, operative findings, and autopsies.

Phlebography.—Phlebograms were performed by a method previously described.[10] The essential features of the technique were: (1) performance of the examination with the patient in a semierect position, (2) the cannulation of any superficial vein on the dorsum of the foot, (3) application of a tight tourniquet around the ankle, (4) the injection of 40 c.c. of 50 per cent Hypaque, and (5) exposure of 14 by 33 inch radiographs of the entire leg before and after exercise.

Technique of Thrombectomy.—The method of thrombectomy used was essentially the same as that described by Mahorner.[25] Anesthesia consisted of either the local infiltration of 1 per cent procaine, the injection of 1.5 per cent Xylocaine into an indwelling epidural catheter, or a general anesthetic. An oblique incision was made over the course of the femoral vein in the groin and extended superiorly and laterally along the inguinal crease for a short distance. The common femoral, greater saphenous, superficial femoral, and deep femoral veins and other branches were mobilized and all but the common femoral vein were occluded. A short longitudinal incision was made in the common femoral vein and the thrombus allowed to extrude. More thrombus was then removed with forceps. Large-bore polyethylene catheters* were cautiously inserted until obstruction was encountered, and any remaining thrombotic material was removed by applying constant suction. An attempt was made to completely evacuate the thrombus from the iliac vein to the level of the inferior vena cava as evidenced by a brisk retrograde flow and the passage of a catheter into the cava itself. The common femoral vein was then occluded and the distal branches similarly evacuated of clots. If competent valves hindered the distal passage of the catheters the thigh was compressed to milk the clots toward the venotomy. An attempt was made to obtain free flow of blood from all of the distal branches. A 5 mg. per cent heparinized

*Sets of five graded sizes of 30 inch long polyethylene endoarterial suction tubes, gas sterilized and doubly packaged in polyethylene bags for immediate operative use, are available from the Klista Company, P. O. Box 715, Nashville, Tenn.

saline solution was then instilled in all branches and the incision in the common femoral vein closed with a continuous everting suture of 5-0 arterial silk. In 3 patients the iliac veins were also exposed through an abdominal incision with manual compression of clots retrograde from the iliac vein through the femoral venotomy. In one instance this was followed by ligation of the inferior vena cava. In most instances postoperative long-acting heparin anticoagulation was instituted.

Methods of Evaluating Results.—The results of therapy were evaluated by means of phlebograms, an assessment of vasospasm, venous pressure determinations, and measurement of leg swelling. *Vasospasm* was evaluated by color and temperature of the leg as well as careful palpation of the peripheral pulses, and oscillometry in some instances. *Serial venous pressure* determinations were made from the level of the right atrium by using a length of sterile plastic tubing attached to a needle in the greater saphenous vein at the ankle and connected to a continuous 2 mg. per cent heparinized saline infusion by a three-way stopcock. *Circumferences* of the ankle, calf, and thigh were measured at the same level over a period of several days.

RESULTS

Origin of the Iliofemoral Thrombosis (Table I).—Analysis of the events preceding the occurrence of the massive edema indicated that the process began in the proximal iliac and femoral veins in 16 of the 27 extremities. The patients either stated or were observed to have sudden swelling, usually accompanied by an aching or pain, involving the whole leg in a matter of hours. Some of these patients even mentioned aching pain in the lower abdomen as the first symptom. Phlebograms of 7 of these extremities also suggested the proximal origin of the thrombus. In 2 instances the obstructive clots were localized to the iliac and proximal femoral or proximal greater saphenous veins without any visible clots distally (Fig. 1). In 5 instances there was extensive proximal obstruction including the superficial femoral vein. However, the popliteal and calf veins, if involved at all, showed only small partially obstructing thrombi (Fig. 2). Examination of the legs added further support to the proximal origin theory in 5 instances. These patients had groin tenderness but no significant calf tenderness, and the Homan's sign was conspicuously absent. The findings at the time of thrombectomy gave the most convincing support in 5 of these 7 extremities. The most organized and adherent thrombi were removed from the iliac vein itself. The distal thrombi were generally friable and more like currant jelly. Histologic studies supported the gross impression that the proximal thrombi were more organized and therefore older.

TABLE I. PROBABLE ORIGIN OF ILIOFEMORAL VENOUS THROMBOSIS IN 27 EXTREMITIES

VEINS	TOTAL NUMBER	HISTORY	EXAMINATION	PHLEBOGRAM	PATHOLOGY
Iliofemoral	16	16	7	7	10
Greater saphenous	3	3	-	1	2
Calf	8	8	6	5	-

There was contributory evidence that the thrombus originated proximally in 7 of the 9 remaining extremities in which the history suggested such an origin. This evidence consisted either of the findings of older thrombi in the iliac vein at the time of operation or autopsy or of the absence of clinical signs of deep calf vein thrombosis. One of these patients did have a phlebogram

which showed extensive obstructive thrombosis of the calf veins, as well as the proximal veins, but at operation and at autopsy the most organized thrombi were found in the proximal iliac veins. The history alone was convincing enough in the remaining 2 patients to include them in this group.

Three patients gave histories of palpable tender cords in the groin prior to the sudden onset of massive swelling, suggesting that the origin was the greater saphenous vein with ascension into the femoral and iliac veins. This concept was supported by phlebography in one and at operation in the other two. The phlebogram of one of these extremities demonstrated extensive obstructive thrombosis of all of the deep veins of the leg, but this patient was not seen until 10 days after the onset of disease.

Fig. 1.

Fig. 2.

Fig. 3.

Fig. 1.—Patient J. R. Localized proximal thrombosis. The upper arrow points to an obstructing thrombus in the iliac vein. The lower arrow points to a nonobstructing thrombus in the femoral vein. Pelvic collaterals are present. The distal femoral, popliteal, and main deep calf veins appear normal.

Fig. 2.—Patient S. H. Extersive proximal thrombosis with nonobstructing distal thrombosis. The iliac and entire femoral vein is obstructed. Numerous pelvic and thigh collaterals are visualized. The popliteal and calf veins contain small thrombi.

Fig. 3.—Patient G. G. Extensive proximal and distal thrombosis. All of the main deep veins are obstructed. Only collaterals are visualized.

The historical evidence suggested that the thrombus originated in the lower leg and progressed proximally in the remaining 8 of the 27 extremities. The distal origin concept was supported by phlebograms in 5 instances (Fig. 3) and by physical findings of deep calf vein thrombosis in the remaining 3 extremities.

The iliac veins have been examined carefully at the time of autopsy in 12 patients without venous disease, in 2 patients with distal thrombosis extending into the iliac veins, and in 5 patients whose massive iliofemoral venous

thrombosis apparently originated in the iliac veins. One of the patients with a massive thrombosis of proximal origin demonstrated a 2 cm. long partial stenosis of the common femoral vein just superior to the iliolumbar branch and beneath the left hypogastric artery. Histologic sections revealed the stenosis to be due to an old organized thrombus with contracture of the wall. No anatomic variations were noted in any of the other specimens examined. Postthrombectomy phlebogram of another patient, who had had proximal onset of disease, gave probable evidence of a tortuous double inferior vena cava.

Thrombectomy.—"Complete" iliac thrombectomies, as evidenced by the passage of a catheter into the inferior vena cava and a brisk retrograde blood flow, was accomplished in 8 of 13 attempted thrombectomies. In the same cases it was also possible to re-establish flow from the femoral vein in 8 and the greater saphenous vein in 7. In 5 cases the iliac vein was not considered patent at the end of the procedure, and despite the fact that one or more of the distal veins was considered reopened these 5 cases have been considered "incomplete" thrombectomies.

Comparative Results of Operative and Nonoperative Treatment (Tables II and III).—The results of therapy have been compared with regard to relief of vasospasm, changes in venous pressure, decrease in swelling, phlebographic changes, complications while in the hospital, and late follow-up findings.

Vasospasm and gangrene: Evidence of vasospasm was present in 12 of the 14 *nonoperated* extremities. This consisted of a mottled reddish cyanosis of the skin particularly in the dependent position and coolness to touch in most instances. In 2 extremities there was a definite recorded decrease in the amplitude of the peripheral pulses. Improvement occurred slowly over a 1- to 5-day period in 6 extremities. There was also transient improvement in 4 extremities following sympathetic blocks. Although diffuse cyanosis disappeared and peripheral pulsations improved in 6 extremities, they still developed localized gangrene within 1 to 6 days after onset of massive swelling. One of these received sympathetic nerve blocks prior to onset of gangrene, and another was receiving an intermittent sympathetic nerve block through an epidural catheter.

Vasospasm was noted in 10 of the 13 *operated* extremities and was accompanied by weak or absent peripheral pulses in 4 extremities. There was an immediate decrease in severity of arterial insufficiency in all extremities in which the thrombectomy was complete. One patient was observed to have a markedly constricted cordlike femoral artery which became a normal pulsating artery during the course of a complete thrombectomy. The skin changes improved immediately in 2 patients and slowly in one patient with incomplete thrombectomies. A markedly spastic femoral artery improved during an incomplete thrombectomy, but the patient subsequently developed an area of superficial gangrene on the medial calf.

Venous pressures: Venous pressures were obtained in 9 extremities with massive venous thrombosis. Measurements varied from 51 to 115 cm. H_2O. Various forms of therapy influenced the pressure favorably. There was a 36 per cent

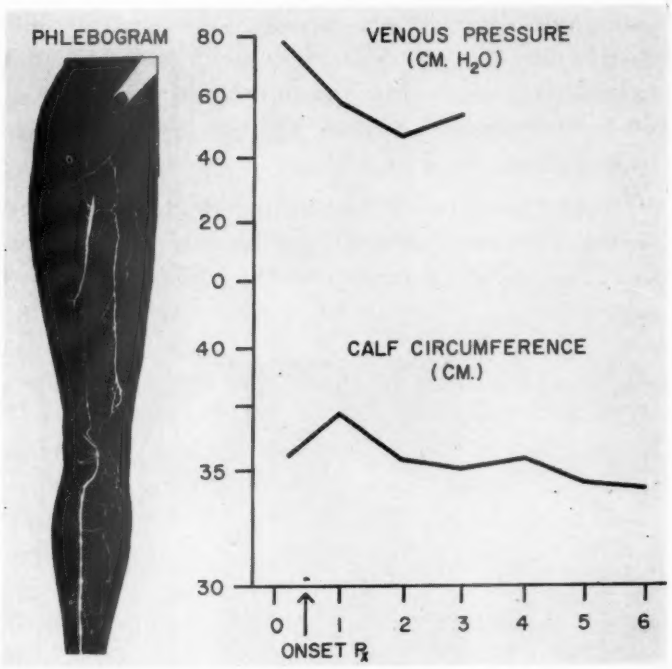


Fig. 4.—Conservative treatment with elevation and heparin.

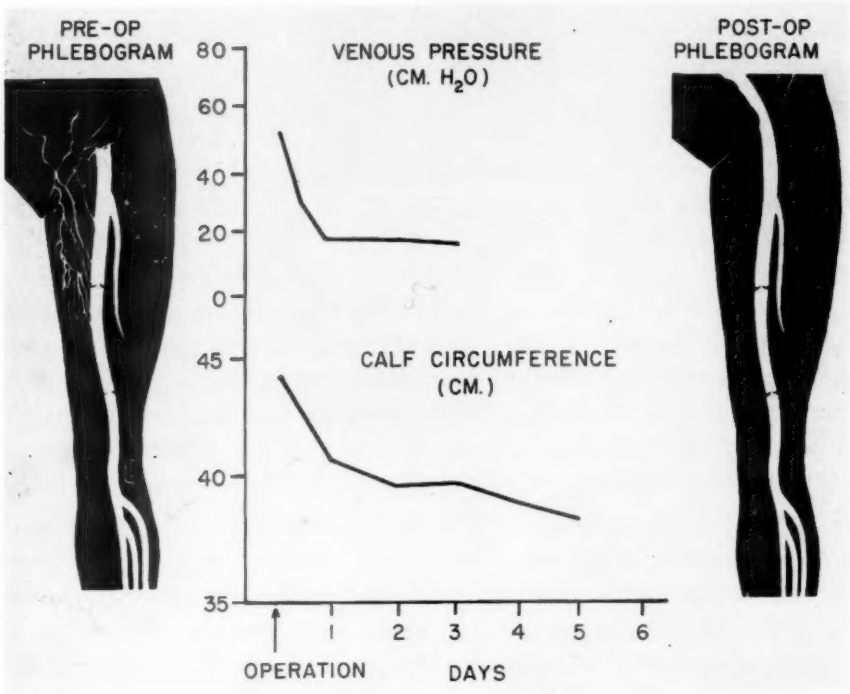


Fig. 5.—Thrombectomy.

decrease toward normal limits in one extremity following one day of bed rest (Fig. 4). A 64 per cent decrease occurred in one extremity immediately following a sympathetic nerve block. There were 51 and 67 per cent decreases in 2 patients immediately following incomplete thrombectomies. The most dramatic immediate decreases of 89 and 100 per cent occurred in 2 patients with complete thrombectomies (Fig. 5).

Swelling: The decrease in swelling was gradual over a period of several days in 6 nonoperated extremities and these patients were discharged with significant edema. The edema persisted to the onset of gangrene in 6 other extremities.

Fig. 6.—Patient M. S. Postthrombectomy phlebogram demonstrating patent greater saphenous and iliac veins. The middle arrow points to the normal position of the femoral vein which is obstructed.

The extremities with complete thrombectomies had marked immediate decreases in swelling ranging from 2 to 5 cm. changes in circumference in the calf or thigh, followed by continual gradual decreases (Fig. 5). The extremities with incomplete thrombectomies showed gradual decreases similar to the nonoperated cases (Fig. 4). However, all of the patients still had some swelling on discharge necessitating the use of elastic support.

Postoperative phlebograms: Phlebograms were obtained after thrombectomy on 7 extremities. The iliac vein was patent at periods of 9 days to 13 months in the 5 extremities in which complete thrombectomies were performed (Fig. 6). The phlebograms suggested that the iliac veins were still blocked 7 weeks and 13 months after incomplete thrombectomies in 2 other extremities. The superficial femoral vein was patent and normal valves were visible 11 days postoperatively in a patient (H. R.) whose preoperative phlebogram indicated that the thrombosis was limited to the iliac and common femoral vein (Fig. 5).

The other 4 extremities in which complete iliac thrombectomies were performed all showed obstruction or various stages of recanalization of the superficial femoral veins. No valves were present. The 2 patients with incomplete thrombectomies still showed obstruction postoperatively. The greater saphenous vein was patent on 6 phlebograms in which an unequivocal statement regarding its patency was possible.

Related complications and mortality: The complications occurring during nonoperative treatment consisted of pulmonary embolization, gangrene, and uremia. Four of the 5 patients with signs and symptoms of pulmonary emboli died and had multiple small emboli demonstrated at autopsy which were felt to be contributory causes of death. Gangrene occurred in 6 extremities 1 to 6 days following onset of massive swelling and was felt to contribute to the death of 4 patients. One patient developed uremia due to renal tubular necrosis possibly secondary to phlegmasia cerulea dolens. There were 5 deaths in this group, and the ultimate cause of death was related to venous thrombosis or its complications in all cases.

Similar related complications were infrequent in the *operated patients.* Only one patient had symptoms suggestive of pulmonary embolization, but radiographs failed to substantiate the diagnosis. Pulmonary emboli were only found in one of 3 patients in this group at autopsy, and it should be noted that these emboli were several days old although the patient died one day postthrombectomy. Therefore, there have been no proved emboli following thrombectomy.

One patient developed a small area of superficial gangrene on the medial calf which was successfully skin grafted. The most frequent complication in the operated group was associated with the wounds. There was delayed wound healing of 5 wounds, and in all instances this was related to early postoperative bleeding and hematomas. The early bleeding occurred in heparinized patients at times when the clotting time ranged from 30 to 90 minutes and frequently necessitated transfusions of 500 to 1,000 c.c. of blood. The difficulty was minimized in later operations by the use of smaller doses of heparin with the careful control of the clotting time within the range of 15 to 25 minutes. One of these patients also had dehiscence of the abdominal wound further complicated by a postoperative intestinal obstruction.

Thrombectomy contributed to the death of one and possibly 2 patients. One patient who was operated upon under general anesthesia died of an aspiration pneumonia one day after operation. Another patient with severe hypertension died of a cerebrovascular accident on the first postoperative day. The other death occurred 8 days postthrombectomy and was due to carcinomatosis, without evidence of pulmonary embolization.

Late morbidity: Persistent edema and in most instances evening aching was present in 7 nonoperated extremities and also in the 9 operated extremities for the first few months after the acute thrombotic episode. It is impossible to compare the two groups objectively since varying types of external support have been used to control the swelling. However, it was felt that control of the swelling in patients with complete thrombectomies was easier in the early

TABLE II.

NAME AGE SEX	INITIATING CAUSES	SIDE	ORIGIN	AGE OF DISEASE	PHLEBOGRAM	TREATMENT
B. A. 54 M	Bed rest, cancer of esophagus	Left	Proximal	1 day	—	Elevation
J. D. 73 M	Spontaneous, previous contralateral thrombosis	Right	Calf	28 days	42 days Iliac blocked Superficial femoral recanalized Greater saphenous open	Elevation
R. F. 55 M	Postoperative lysis adhesions	Left	Proximal	1 day	—	Elevation, 1 sympathetic block, Tromexan
G. G. 63 M	Bed rest, cancer of lung	Right	Calf	1 day	1 day Iliac blocked Superficial femoral blocked Greater saphenous blocked	Elevation, continuous sympathetic block, heparin
M. H. 70 F	Postoperative	Left	Proximal	1 day	—	Elevation, heparin and Tromexan
J. J. 53 M	Bed rest, cardiac	Left	Proximal	21 days	35 days Iliac blocked Superficial femoral blocked Greater saphenous blocked	Elevation, heparin and Coumadin
E. M. 57 F	Bed rest, ulcerative colitis	Left	Proximal	1 day	—	Elevation, sympathetic blocks
W. O. 54 M	Bed rest, pneumonia	Right	Calf	3 days	—	Elevation, Dicumarol
	Contralateral thrombosis	Left	Proximal	1 day	—	Elevation, Dicumarol
E. R. 25 F	Bed rest, synergistic gangrene of breast	Left	Calf	1 day	—	Elevation, heparin and Tromexan
A. S. 78 M	Spontaneous	Left	Proximal	1 day	6 days Iliac blocked Superficial femoral blocked Greater saphenous open	Elevation, heparin and Dicumarol
S. T. 31 F	Spontaneous, 8 months pregnant	Right	Great saphenous	1 day	19 days Iliac blocked Superficial femoral blocked Greater saphenous blocked	Elevation, heparin
L. P. 62 M	Bed rest, cancer of lung	Left	Proximal	1 day	—	Elevation, Coumadin
	Contralateral thrombosis	Right	Proximal	1 day	—	Elevation, Coumadin

VASOSPASM AND GANGRENE	VENOUS PRESSURES	SWELLING	RELATED COMPLICATIONS	LATE FOLLOW-UP
—	—	—	Pulmonary emboli	Died 1 day, pulmonary emboli, carcinomatosis
None	—	Gradual decrease	None	24 months—swelling uncontrolled with Ace bandages
Decreased pulses, progressed to gangrene 2 days	—	Persisted	Gangrene leg, uremia	Died 14 days, peritonitis and renal tubular necrosis
Progressed to gangrene 1 day	67% decrease sympathetic block	Persisted	Gangrene, pulmonary emboli	Died 15 days, pulmonary emboli, carcinomatosis
Decreased 1 day	—	Gradual decrease	None	4 years—asymptomatic swelling first 4 months
Decreased pulses, improved 1 day	—	Gradual decrease	None	8 months—swelling controlled with elastic stocking
Decreased 4 days	—	Gradual decrease	None	30 months—asymptomatic, swelling first 4 months
Progressed to gangrene 6 days	—	Persisted	Gangrene, pulmonary emboli	Died 23 days, pulmonary emboli, thrombosis inferior vena cava
Progressed to gangrene 1 day	—	Persisted	Gangrene, pulmonary emboli	
Decreased slowly	—	Prolonged	Pulmonary emboli	26 months—swelling uncontrolled
Decreased over 5 days	—	Gradual decrease	None	8 months—swelling uncontrolled with Ace bandage
Decreased over 5 days	36% 1 day	Gradual decrease	None	3 months—swelling controlled with elastic stockings
Progressed to gangrene 4 days	—	Persisted	Gangrene, pulmonary emboli	Died—16 days, pulmonary emboli, carcinomatosis
Progressed to gangrene 2 days	—	Persisted	Gangrene, pulmonary emboli	

TABLE III.

NAME AGE SEX	ONSET	SIDE	SOURCE	AGE OF DISEASE	PHLEBOGRAM	OPERATION
L. B. 70 M	Spontaneous	Left	Calf	9 days	Iliac blocked Superficial femoral blocked Greater saphenous open	Complete
V. D. 61 M	Postoperative, cancer of pancreas, contralateral thrombosis	Right	Calf	1 day	Iliac blocked Superficial femoral blocked Greater saphenous open	Complete
	Postoperative, cancer of pancreas	Left	Calf	12 days	Iliac blocked Superficial femoral blocked Greater saphenous blocked	Incomplete
W. E. 62 M	Spontaneous	Left	Proximal	1 day	Iliac blocked Superficial femoral blocked Greater saphenous open	Incomplete
S. H. 49 F	Postoperative	Left	Proximal	4 days	Iliac blocked Superficial femoral blocked Greater saphenous open	Incomplete
M. I. 48 F	Postinjury, bed rest	Left	Proximal	1 day	—	Complete
C. P. 42 M	Postoperative	Left	Proximal	1 day	Iliac blocked Superficial femoral blocked Greater saphenous open	Incomplete
J. R. 71 M	Spontaneous	Left	Proximal	5 days	Iliac blocked Superficial femoral open Greater saphenous open	Complete + inferior vena caval ligation
H. R. 20 M	Spontaneous, previous contralateral thrombosis	Left	Proximal	5 days	Iliac blocked Superficial femoral open Greater saphenous blocked	Complete
J. S. 40 M	Spontaneous	Right	Great saphenous	10 days	Iliac blocked Superficial femoral open Greater saphenous blocked	Complete
	Postoperative, contralateral thrombosis	Left	Great saphenous	1 day	—	Incomplete

VASOSPASM GANGRENE	VENOUS PRESSURES	SWELLING	PHLEBOGRAM	RELATED COMPLICATIONS	LATE FOLLOW-UP
None	—	—	—	Aspiration pneumonia	Died 1 day, pneumonia and old pulmonary emboli
None	—	Gradual decrease	—	None	Died 8 days, carcinomatosis
Immediate decrease	—	Immediate decrease	—	None	
—	67% decrease immediately	No change	—	Cerebrovascular accident	Died 1 day, cerebrovascular accident
Gradual decrease	—	Gradual decrease	7 wk. Iliac blocked Superficial femoral blocked Greater saphenous open	Bleeding, hematoma, delayed healing	5 months—swelling uncontrolled with Ace bandage
Absent pulses, immediate improvement	100% decrease immediately	Immediate decrease	6 wk. Iliac open Superficial femoral blocked Greater saphenous open	Bleeding, hematoma, delayed healing	2 months—swelling controlled with elastoplast boot
Absent pulses, improved superficial gangrene	51% decrease immediately	Gradual decrease	—	Superficial gangrene grafted, wound infection	5 months—swelling controlled, graft site healed
Decreased pulses, immediate improvement	—	—	—	None	4 months—swelling controlled with Aeropulse boot
Immediate decrease	89% decrease immediately	Immediate decrease	11 days—9 months Iliac open Superficial femoral open Greater saphenous	None	9 months—asymptomatic, minimal swelling with elastic stocking
Immediate decrease	—	Gradual decrease	13 months Iliac recanalized Superficial femoral recanalized Greater saphenous open	Hematoma, dehiscence, intestinal obstruction	13 months—swelling controlled with elastic stocking
Decreased pulses, immediate improvement	—	Gradual decrease	13 months Iliac blocked Superficial femoral recanalized ± Greater saphenous open	None	13 months—swelling controlled with elastic stocking

TABLE III.

NAME AGE SEX	ONSET	SIDE	SOURCE	AGE OF DISEASE	PHLEBOGRAM	OPERATION
M. S. 71 F	Postoperative, cancer of rectum	Left	Proximal	1 day	—	Complete
N. Z. 70 M	Spontaneous	Right	Proximal	5 days	Iliac blocked Superficial femoral blocked ± Greater saphenous open	Complete

postoperative period than in either the nonoperated extremities or the ones with incomplete thrombectomy. The most objective comparison was in Patient H. R., who had had a right massive iliofemoral thrombosis 7 years prior to admission for the same condition of the left leg. Following a complete thrombectomy, the swelling decreased rapidly in the left leg and has remained 3 to 4 cm. smaller in circumference at the calf and thigh levels through an 8-month follow-up period. No patient in either group has developed stasis changes or ulceration in this short follow-up period.

DISCUSSION

The thrombus which occludes the iliac and/or the common femoral vein may originate from distal deep vein thrombosis or from thrombosis in the greater saphenous vein or may arise primarily in iliofemoral veins. There was convincing evidence that the thrombus originated in the calf veins in only 8 of the 27 extremities in this series. In the remaining extremities, however, the apparent origin of the thrombi was proximal and usually within the iliofemoral veins. The reason for formation of thrombi in these veins may be the presence of congenital[29] or acquired[12, 28] partially obstructing lesions in the iliac veins which are frequently found in the left common iliac vein at the site where the right iliac artery crosses it. These have also been described in the iliac veins at the site where the internal iliac arteries cross the vein.[28] The fact that these lesions are predominantly on the left side and most cases of iliofemoral venous thrombosis occur on the left, as was once again demonstrated in this series, suggested a causal relationship. Such a lesion was demonstrated at autopsy in Patient W. E. of this series. The inguinal ligament is also a possible causal factor for primary thrombosis in the iliofemoral region.[2] Thrombosis in the smaller pelvic veins may spread into the iliac veins.[5] In addition thrombosis of the greater saphenous vein may ascend into the common femoral vein as was demonstrated in 3 of the 27 extremities in this series.[18, 21]

Bauer[4] theorized that all thrombi which occluded the femoral vein originated from the deep calf veins on the basis of phlebographic studies. However, he admitted that his method of phlebography did not include radiographs of

(CONT'D)

VASOSPASM GANGRENE	VENOUS PRESSURES	SWELLING	PHLEBOGRAM	RELATED COMPLICATIONS	LATE FOLLOW-UP
None	—	Immediate decrease	9 days Iliac open Superficial femoral blocked Greater saphenous open	None	1 month—swelling uncontrolled
Immediate decrease		Immediate decrease	3 wk. Iliac open Superficial femoral recanalized Greater saphenous open	Bleeding, hematoma, delayed healing	2 months—swelling controlled with Ace bandages

the upper thigh and pelvis. Phlebograms of Patients J. R. and H. R. (Figs. 1 and 5) show convincing evidence that there need not be any significant distal deep vein thrombosis in the presence of proximal disease. The concept that most thrombi originate distally has also been based on autopsy studies. A review of one of the often-quoted articles on this subject by Hunter, Sneeden, Robertson, and Snyder[20] does indeed indicate that in almost all instances thrombi were found in calf veins and not in the iliofemoral veins. However, Hunter quotes Rossle[32] as finding thrombotic involvement of the femoral or hypogastric veins in the absence of thrombosed leg veins in 7 of 133 instances of deep venous thrombosis and also described similar cases in his own series. In a later report Bauer[3] also mentioned 7 cases which he felt started in the pelvic or upper femoral veins. It is not our contention that the iliofemoral veins are the source of all thrombi in venous thrombosis of the leg, but only that they are the probable source of thrombi in the majority of cases of iliofemoral venous thrombosis.

The introduction of catheters or sucker tips into the iliac vein for removal of thrombi without proximal control of the vessel was initially of some concern to us, and the first thrombectomy on Patient J. R. was performed after the inferior vena cava was ligated. However, the venous pressure in the iliac veins is positive, and during the time that the venotomy incision is open or a suction catheter is being cautiously introduced there should be no danger of embolization.[21] The possible danger is in breaking off clots which are not removed by suction or flushing before closure of venotomy and restoration of normal circulation. With reasonable caution, however, there seems to be little to fear as attested by the lack of embolization in our series as well as those of Läwen,[22] Fontaine,[16] Mahorner,[26] and Edwards.[11] Furthermore, iliac thrombectomy was performed by Allen,[1] Gage,[17] deTakats,[7] Szilagyi,[33] Lowenberg,[24] Kulenkampff,[21] Mavor,[27] Glover,[18] and others in conjunction with ligation of the femoral or saphenous veins, without apparent fear of embolization, and in many instances the indication for thrombectomy was to prevent further embolization.

The ultimate aim of thrombectomy as described is to remove all thrombi from the iliac, femoral, and greater saphenous system to restore normal deep venous return. At the operating table two criteria were used to confirm that

patency had been re-established. The first criterion was the presence of a brisk flow of blood from the open venotomy, when all but the branch in question was occluded. The second was the passage of a catheter a significant distance into the vein, and in the iliac vein this meant passing it into the inferior vena cava. Using a combination of these criteria it was possible to re-establish iliac patency in 8 of 13 thrombectomies. One failure was due to a stenosis of the common iliac vein due to an older thrombotic process as established at subsequent autopsy. The reason for the other failures became more obvious following a series of experiments in the autopsy room. The common femoral vein was exposed bilaterally in 12 patients post mortem and various types of catheters were passed into the iliac vein through a femoral venotomy. All sizes of polyethylene catheters and glass tubes routinely met an obstruction which could not be passed by any maneuver approximately 15 cm. above the inguinal ligament. The obstruction proved to be the bony wall of the pelvis

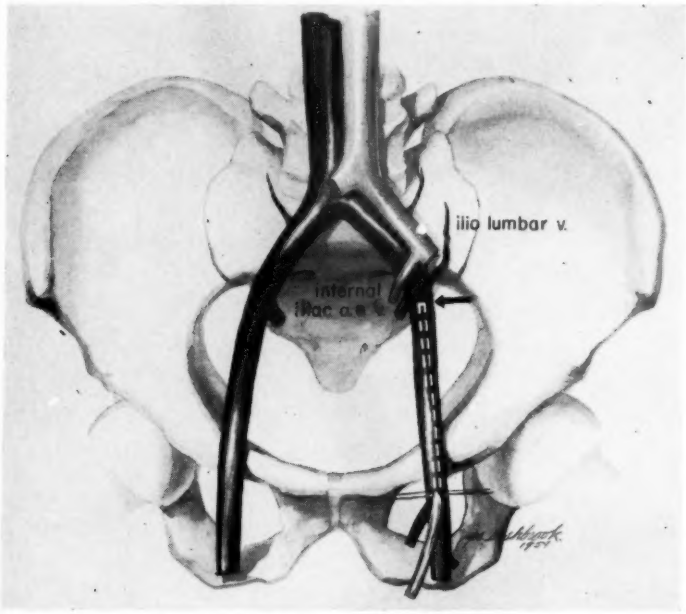


Fig. 7.—A rigid catheter has been introduced into the iliac vein via a common femoral venotomy. The catheter has hit the posterior wall of the pelvis at the point where the vein angulates anteriorly and medially over the sacrum. The vein is immobilized at this point by iliolumbar and internal iliac branches as well as the overlying internal iliac artery.

near the sacroiliac joint at which point the veins angled medially and anteriorly to pass over the sacrum. In addition the vein was held posteriorly at this point by the iliolumbar and internal iliac branches of the vein and also the overlying hypogastric artery (Fig. 7). This difficulty has *not* been previously mentioned and may be falsely interpreted as thrombotic occlusion. Only soft rubber catheters easily passed this angle into the vena cava. Consequently, it is likely that there were adherent thrombi in common iliac veins above this angle in the 4 failures. Unfortunately, soft rubber catheters have not been as successful as rigid polyethylene tubings in coring out adherent thrombi. Three patients were also explored abdominally, but the tremendous number of

dilated collaterals made exposure of the iliac veins difficult. The main value was that it made it possible manually to express clots through the femoral venotomy. However, laparotomy also increases the hazards of the procedure as in Patient J. S., who developed intra-abdominal bleeding wound dehiscence, and intestinal obstruction following such a procedure.

Following thrombectomy there is generally an immediate relief of vasospasm in our experience as well as others.[11, 16] This improvement was even noted following an incomplete thrombectomy in this series and apparently occurs even if the femoral vein is ligated following thrombectomy.[6, 13] It seems logical to us that the mottled cyanosis so frequently observed in patients with massive iliofemoral venous thrombosis is usually a combination of arteriospasm and venospasm as postulated by Ochsner and DeBakey.[31] This was demonstrated by temporary improvement in pulses in Patients R. F., G. G., and E. M. in this series following sympathetic blocks. This was further supported by a marked decrease in the venous pressure of the involved extremity of G. G. with the block, as has been previously demonstrated by Edwards.[11] However, the concept that some of this appearance and certainly the eventual gangrene is due to massive occlusion of the small veins, as postulated by DeBakey and Ochsner,[6] and Haimovici,[19] was suggested by the occurrence of gangrene in 2 of the same extremities (G. G. and R. F.) following temporary improvement with the blocks. Furthermore, one of these patients (G. G.) had extensive thrombosis of all the major deep veins of the leg as demonstrated by phlebography (Fig. 3). On the basis of these cases, we cannot agree with Haimovici[19] that localized iliofemoral venous thrombosis with vasospasm can be differentiated from massive venous thrombosis by sympathetic blocks. However, phlebograms can give an accurate picture of the extent of the disease and can aid in differentiation.[9, 15] The mechanism by which thrombectomy decreases vasospasm is not clear. The removal of obstructing thrombi may reflexly release the spasm.[6] In addition the thrombectomy may prevent gangrene secondary to the massive thrombotic occlusion of smaller veins by opening the major venous channels.[23]

The related complications observed in the nonoperated cases were particularly severe because they contributed to the 5 deaths in this group. Embolization and gangrene were the usual complications. However, one patient (R. F.) also had renal failure which was possibly secondary to the sudden massive pooling of blood in the extremities and peripheral circulatory collapse.[30] The related complications in the operated group were temporarily disabling, but complications related to venous thrombosis were not the cause of death in any of these 3 patients. However, there were 2 deaths in the immediate postoperative period which must be attributed to the operative procedure. The aspiration pneumonia was probably secondary to the use of a general anesthetic in a poor-risk patient, and therefore, local or epidural anesthesia is now the procedure of choice.

Mahorner reported little or no swelling in the late postoperative period in 8 of the 10 patients in whom iliofemoral thrombectomies were performed. There was swelling of moderate degree to periods up to 13 months postoper-

atively in 8 of 9 patients in our series. It was felt that this swelling was easier to control in operated as opposed to nonoperated patients during this short period. The results were best explained by postoperative phlebograms. Patient H. R. (Fig. 5) whose disease was localized to the iliac and common femoral veins by preoperative phlebograms had normal-appearing deep veins postoperatively including valves in the superficial femoral vein and is the single excellent result with only minimal edema. The remaining patients demonstrated varying degrees of recanalization of their superficial femoral vein, suggesting that they rethrombosed following the thrombectomy. Mahorner published two postoperative phlebograms, both of which demonstrated patent iliac veins and also a beautiful vein with valves which in our experience correspond to the course of the profunda femoris vein.[26] The superficial femoral veins, however, have the appearance of being partially obstructed in one instance and partially recanalized in the other instances. It seems, therefore, that in many instances, particularly when there is extensive involvement of the distal veins, the result of the operation is to convert an iliofemoral venous thrombosis into a femoral vein thrombosis. These veins do recanalize but leave their scars in the presence of valveless poorly functioning veins.[8, 10] The possibility that this will be the end result in these cases of more extensive thrombosis is also suggested by the results of Fontaine.[16] After a follow-up period of 2 to 6 years 10 of 22 thrombectomized extremities, as compared to 6 of 15 nonoperated ones, had either persistent swelling or ulcers of the lower leg. Therefore, there seems to be little to gain by performing thrombectomy in the presence of extensive distal thrombosis unless uncontrollable vasospasm is present.

SUMMARY

Since iliofemoral venous thrombosis usually originates in the proximal veins, there is a time early in the disease, before extensive distal thrombosis occurs, when the "ideal" thrombectomy should be followed by an ideal result of complete return of normal anatomy and function. If extensive distal thrombosis occurs or if the thrombosis has ascended from the calf veins, thrombectomy is indicated in the presence of severe vasospasm or a pregangrenous appearance to decrease early morbidity. There is no clear-cut indication for performing thrombectomy in the absence of vasospasm or impending gangrene and with extensive distal thrombosis, since thrombectomy has little chance of decreasing late morbidity under those circumstances.

CONCLUSIONS

1. Acute iliofemoral venous thrombosis was observed and treated in 27 extremities of 23 patients during a 4-year period.

2. There was evidence that the thrombosis originated proximally in the iliac, common femoral, or greater saphenous veins in 19 of the extremities.

3. Thrombectomy without distal vein ligation was performed on 13 extremities of which 8 were considered complete thrombectomies. Rigid catheters introduced into the iliac vein via the femoral vein could not be passed beyond

the acute angulation of the vein on the posterior pelvic well and prevented complete thrombectomies in some cases.

4. Operated cases showed less early morbidity and mortality, particularly with regard to those problems associated with swelling, vasospasm, and gangrene. The later morbidity of chronic swelling remained a problem in most instances.

5. The best results were obtained in patients whose thrombosis was localized to the iliac and femoral veins as determined by preoperative phlebograms. Postoperative phlebograms demonstrated continued thrombosis or recanalization of the distal femoral veins in patients with postphlebitic problems.

6. Thrombectomy is a valuable adjunct in the management of iliofemoral venous thrombosis in those cases in which the process is localized proximally, or in those in which there is evidence of vasospasm or iminent gangrene.

We wish to express our thanks to Dr. W. J. M. Scott for his support of this study, to Drs. William Clark and Harold Bales who performed some of the thrombectomies, to Dr. S. M. Rogoff who has been of continual help in the performance and interpretation of the phlebograms, and to Mr. Howard Netter who furnished valuable assistance in this study as a Medical Student Fellow of the National Foundation for Infantile Paralysis.

REFERENCES

1. Allen, A. W., Linton, R. R., and Donaldson, G. A.: Thrombosis and Embolism, Review of 202 Patients Treated by Femoral Vein Interruption, Ann. Surg. **118**: 728, 1943.
2. Aschoff, L.: Lectures on Pathology, New York, 1924, Paul B. Hoeber, Inc., p. 264.
3. Bauer, G.: Sequels of Postoperative Venous Thrombosis, Tr. Cong. Soc. Internat. Chir., Paris, 1951, p. 451.
4. Bauer, G.: A Venographic Study of Thrombo-Embolic Problems, Acta chir. scandinav. **84** (Suppl. 61): 1, 1940.
5. Collins, C. G., MacCallum, E. A., Nelson, E. W., Weinstein, B. B., and Collins, J. H.: Suppurative Pelvic Thrombophlebitis, SURGERY **30**: 298, 1951.
6. DeBakey, M. E., and Ochsner, A.: Phlegmasia Cerulea Dolens and Gangrene Associated With Thrombophlebitis, SURGERY **26**: 16, 1949.
7. deTakats, G., and Jesser, J. H.: Pulmonary Embolism, J.A.M.A. **114**: 1415, 1940.
8. DeWeese, J. A.: Functional Popliteal Phlebography in the Patient With a Complicated Varicose Vein Problem, SURGERY **44**: 390, 1958.
9. DeWeese, J. A., and Rogoff, S. M.: Clinical Uses of Functional Ascending Phlebography of the Lower Extremity, Angiology **9**: 268, 1958.
10. DeWeese, J. A., and Rogoff, S. M.: Functional Ascending Phlebography of the Lower Extremity of Serial Long Film Technique; Evaluation of Anatomic and Functional Detail in 62 Extremities, Am. J. Roentgenol. **81**: 841, 1959.
11. Edwards, W. S.: Observations on the Pathogenesis and Management of Massive Venous Occlusion, SURGERY **43**: 153, 1958.
12. Ehrich, W. E., and Krumbhaar, E. B.: A Frequent Obstructive Anomaly of the Mouth of the Left Common Iliac Vein, Am. Heart J. **26**: 737, 1943.
13. Ellis, J. T., and Windham, S. W.: Acute Massive Venous Occlusion in the Lower Extremity, Ann. Surg. **135**: 262, 1952.
14. Felsenreich, F.: Die "ideale Thrombektomie" als Behandlungsmethode der blanden venosen Thrombosen (Phlebothrombosen), Bruns' Beitr. klin. Chir. **192**: 7, 1956.
15. Fine, J., Frank, H. A., and Starr, A.: Recent Experiences With Thrombophlebitis of the Lower Extremity and Pulmonary Embolism, Ann. Surg. **116**: 574, 1942.
16. Fontaine, R.: Remarks Concerning Venous Thrombosis and Its Sequelae, SURGERY **41**: 6, 1957.
17. Gage, M.: Thrombo-Embolic Phenomenon—A Local Process With Regional Spread and Systemic Complications, Ann. Surg. **137**: 577, 1953.
18. Glover, W. J., Vaughn, A. M., and Caserta, J. A.: Venous Thrombectomy in the Management of Acute Venous Thrombosis of the Saphenous System, Am. J. Surg. **93**: 798, 1957.
19. Haimovici, H.: Gangrene of the Extremities of Venous Origin, Circulation **1**: 225, 1950.

20. Hunter, W.C., Sneeden, V. D., Robertson, T. O., and Synder, G. A. C.: Thrombosis of the Deep Veins of the Leg, Arch. Int. Med. **68**: 1, 1941.
21. Kulenkampff, D.: Die Verhutung schwerer und todlicher Embolien durch Ausraumung der Vena Iliaca, Arch. klin. Chir. **193**: 727, 1938.
22. Läwen, A.: Weitere Erfahrungen über operative Thrombenentfernung bei Venenthrombose, Arch. klin. Chir. **193**: 723, 1938.
23. Linton, R. R.: Peripheral Vascular Disease, New England J. Med. **260**: 370, 1959.
24. Lowenberg, E.: Femoral Vein Thrombectomy and Regional Heparinization in Lieu of Vena Cava Ligation for Thrombophlebitis and Pulmonary Embolism, Am. Surgeon **21**: 1038, 1955.
25. Mahorner, H.: A New Method of Management for Thrombosis of Deep Veins of the Extremities, Am. Surgeon **20**: 487, 1954.
26. Mahorner, H., Castleberry, J. W., and Coleman, W. O.: Attempts to Restore Function in Major Veins Which Are the Site of Massive Thrombosis, Ann. Surg. **146**: 510, 1957.
27. Mavor, G. E.: Personal communication.
28. May, R., and Thurner, J.: The Cause of the Predominantly Sinistral Occurrence of Thrombosis of the Pelvic Veins, Angiology **8**: 419, 1957.
29. McMurrich, J. P.: The Occurrence of Congenital Adhesions in the Common Iliac Veins and Their Relation to the Thrombosis of the Femoral and Iliac Veins, Am. J. Med. Sc. **135**: 342, 1908.
30. Morgan, E. H., Allen, E. V., and MacCarty, C. S.: Acute Peripheral Circulatory Failure Caused by Acute Venous Thrombosis, Proc. Staff Meet. Mayo Clin. **23**: 425, 1948.
31. Ochsner, A., and DeBakey, M. E.: Thrombophlebitis; The Role of Vasospasm in the Production of the Clinical Manifestations, J.A.M.A. **114**: 117, 1940.
32. Rossle, R.: Über die Bedeutung und die Entstehung der Wadenvenenthrombosen, Virchows Arch. path. Anat. **300**: 180, 1937.
33. Szilagyi, B. E., and Alsop, J. F.: Early and Late Sequelae of Therapeutic Vein Ligation for Thrombosis of Veins of Lower Limbs, Arch. Surg. **59**: 633, 1949.

Discussion

DR. EUGENE OSIUS (Detroit, Mich).—While I have seen a number of these cases, I still find them terrifying and not too infrequent so far as occurrence is concerned. We have come to the conclusion that thrombectomy is the logical procedure to carry out, just as embolectomy is for arterial occlusions.

Some years ago we reported a small series of these cases and we had somewhat the same results as Dr. DeWeese, although we approached the thrombus through the superficial femoral vein with the idea that this could then be sacrificed by ligation without serious untoward results, and that there would then be fewer embolic phenomena from the femoral than if the vessel were left open or patent.

Generally speaking, we did not use anticoagulants because we found there is often difficulty with the anticoagulants producing hematomas in the wound. We have found the use of a slightly curved gallstone forceps very helpful in the extraction of a thrombus. The gallstone forceps can be put into the vein a surprisingly long distance and very frequently with excellent results so far as the extraction of the thrombus is concerned. Lacquered braided catheters, together with suction, have also been very helpful, although on the left side, because of the normal twist of the iliac vein, these are sometimes not as helpful as we would like. Our results were satisfying, and most of our patients did very well.

We do feel, however, as previous essayists have said, that the patient must have a very definite follow-up from the standpoint of support of the extremity, instruction as to the care of the leg, and, as Dr. Joseph Luke of Montreal has said, "a new way of life."

If these dicta are followed, the results are often gratifying, and the patients progress well, and return to active life. A careful evaluation of every patient, together with a knowledge of the condition, namely, that there is such a thing as acute massive venous occlusion, will lead to the correct diagnosis and the proper treatment.

DR. GORDON A. DONALDSON (Boston, Mass.).—I would like to introduce a word of caution that is derived from our experience with these people with thrombosis at this level.

We are in the process of reviewing our femoral vein series, approximately 4,000 cases at Massachusetts General Hospital. In approximately 15 per cent of these individuals thrombosis was found at the femoral level, and in 40 per cent of these the process had extended into the iliofemoral level.

We have found that in our "bad news" results, the patients who developed massive embolism following vein interruption, a great number fell into this group who had iliofemoral thrombosis, and we felt our procedure had done these patients little good in eliminating the danger of fatal embolism.

We wonder whether we are not invading a nonsurgical area in this particular type of disease, although the authors have presented effective operative therapy. It seems to us that operative manipulation of these veins may carry a high mortality from massive embolism, resulting from secondary thrombus formation. We are looking forward to the appearance of a fibrinolysin which will be effective and not as toxic as the present ones on the market.

EXPERIMENTAL SHAM CORONARY ENDARTERECTOMY WITH AND WITHOUT CORONARY ARTERY PERFUSION

CHARLES FINEBERG, M.D., NICHOLAS P. FORIS, M.D., AND JOHN H. GIBBON, JR., M.D., PHILADELPHIA, PA.

CORONARY endarterectomy has been successfully performed for the relief of angina pectoris in human beings who have had completely occluded coronary vessels.[1, 3, 5] Temporary occlusion of diseased coronary vessels still supplying blood to the myocardium could conceivably result in myocardial infarction, fibrillation, and cardiac arrest.

In studying the problem of revascularization of the dog's heart we have performed endarterectomy with and without coronary artery perfusion on dogs with patent relatively normal coronary vessels.

Most of the experimental work done on revascularization of the myocardium of the dog is based on deliberate occlusion of the left anterior descending coronary artery, as a challenge to the procedure performed.[2, 7, 8] Experiments reported in the literature, as well as our control series have shown that 60 to 90 per cent of the experimental animals will survive acute ligation of this vessel distal to the septal artery if a high oxygen saturation is provided via an endotracheal tube.[4, 6] We believe that occlusion of the circumflex coronary artery just distal to its bifurcation from the left main coronary artery is a more critical test of the procedures being performed for the following reasons: (1) In over 100 surgical procedures no dog with complete arterial occlusion at this point has survived. (2) The hearts of 85 per cent of the animals fibrillated after this artery was occluded for more than 3 minutes. In the remaining 15 per cent significant collateral circulation between the left anterior descending artery and the descending branches of the circumflex coronary artery has been demonstrated. (3) The predominant artery in the dog's heart, unlike the human heart, is the left circumflex coronary artery. This is shown in Fig. 1 which is a photograph of a plastic injection cast of the normal dog's heart. We estimate that the left circumflex coronary artery supplies approximately 60 per cent of the ventricular mass, the left anterior descending coronary artery 25 per cent, and the right coronary artery 15 per cent.

We have been able to maintain normal electrocardiographic patterns and cardiac action in dogs during occlusion of the circumflex coronary artery for a period of 2 hours with distal coronary artery perfusion supplying as little as 10 ml. of blood per minute.

METHODS

Mongrel dogs weighing between 15 and 25 Kg. were utilized. With an endotracheal tube in place a left intercostal thoracotomy incision was made with the dog under light

Read at the Thirteenth Annual Meeting of the Society for Vascular Surgery, Atlantic City, N. J., June 7, 1959.

sodium Pentothal anesthesia. The pericardium was opened anterior to the phrenic nerve and the left auricular appendage was retracted for visualization of the circumflex coronary artery. The circumflex coronary artery was dissected free from enveloping fibrofatty tissue. The branches coming off the coronary vessel at this point were usually left intact. Two stay sutures were placed about the coronary vessel for traction. A small transverse arteriotomy was made in the vessel close to its origin. The arteriotomy was closed at the conclusion of the procedure with 2 or 3 sutures of 6-0 arterial silk.

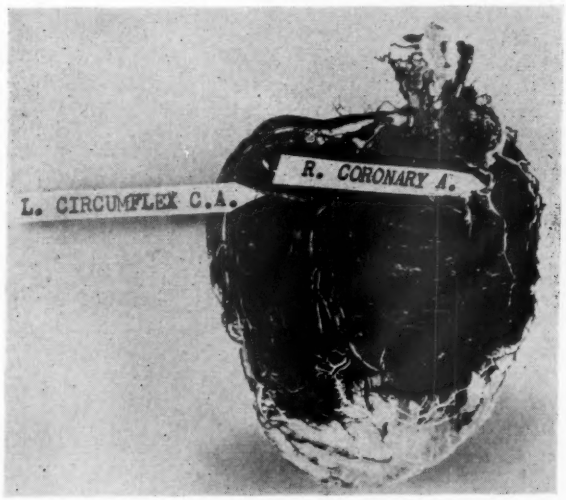


Fig 1.—Plastic injection cast of normal dog heart.

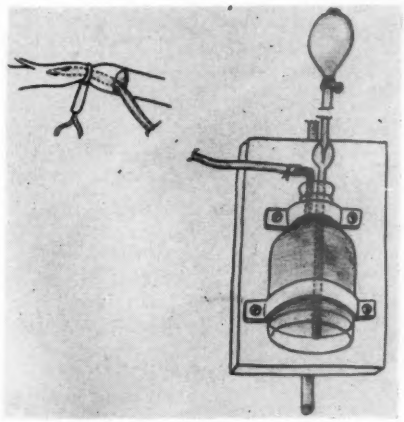


Fig. 2.

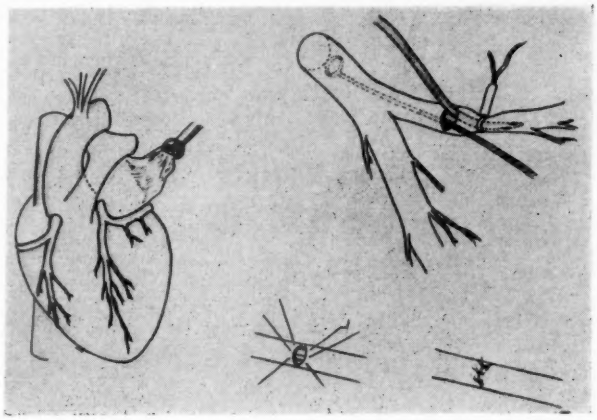


Fig. 3.

Fig. 2.—Perfusion bottle system.
Fig. 3.—Method of coronary endarterectomy.

Group 1. Sham Coronary Artery Endarterectomy With Distal Coronary Artery Perfusion.—A simple pressure bottle system containing heparinized blood freshly drawn from the artery of a donor animal was used for perfusion of the coronary artery (Fig. 2). The saturation of the blood varied from 80 to 90 per cent. A cardiac catheter was placed through the arteriotomy for perfusion of the distal coronary vessels. A Longmire-Cannon coronary artery curette was then passed through the arteriotomy in a proximal direction into the main left coronary artery and at times directly through the coronary ostia into the aorta (Fig. 3). Periods of 8 to 15 minutes were utilized in performance of the sham coronary endarterectomy in this group. Blood (12 to 23 ml. per minute) was supplied during these procedures. Table I presents the results obtained. Four of the 5 dogs survived the operative procedure.

All of these animals died in the immediate postoperative period. Postmortem examination revealed thrombosis at the site of arteriotomy and in the distal coronary vessel, and myocardial infarction in all animals. The proximal portion of the artery was patent in all cases. Fibrillation was not observed during the performance of the endarterectomy except in Dog 826 and in this case it was due to a technical error in closing the arteriotomy.

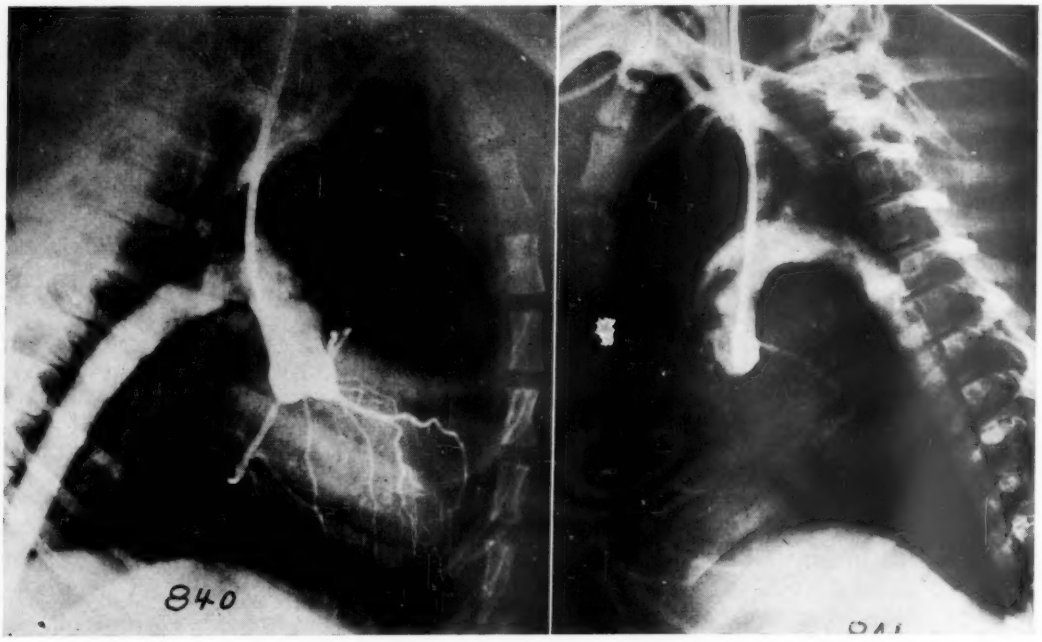


Fig. 4. Fig. 5.

Fig. 4.—Postoperative coronary arteriogram.
Fig. 5.—Postoperative coronary arteriogram.

TABLE I. ENDARTERECTOMY WITH CORONARY ARTERY PERFUSION

DOG NUMBER	PERFUSION (MIN.)	BLOOD (C.C./MIN.)	FIBRILLATION	SURVIVAL	RESULTS
822	8	23	–	11 days	Thrombosis, infarction
823	11	22	–	2 days	Thrombosis, infarction
824	15	16	–	10 hours	Thrombosis, infarction
825	12	12	–	12 hours	Thrombosis, infarction
826	10	14	+	30 min.	Technical error in closure

Group 2. Sham Coronary Artery Endarterectomy Without Coronary Artery Perfusion.—Periods of 1 to 3 minutes of occlusion were utilized for performing sham endarterectomy in this group. Simple digital control of the arteriotomy, which allowed blood to pass into the distal coronary tree, permitted repetition of 3-minute periods of endarterectomy. The 3-minute period without perfusion was not exceeded in order to avoid fibrillation. Table II shows the results of this procedure. All 13 animals survived the operation. Fibrillation was not noted in any. One animal, Dog 836, died after 5 days; postmortem examination revealed a myocardial infarction and thrombosis at the site of arteriotomy. This is the only dog of this series which showed thrombosis at the operative site. Dogs 829 and 830 died of mal-

TABLE II. CORONARY ENDARTERECTOMY

DOG NUMBER	SURVIVAL (MONTHS)	RESULTS
827	6	Alive, x-ray patent
828	6	Alive, x-ray patent
829	3½	Malnutrition, artery patent
830	1	Malnutrition, artery patent
833	6	Alive, x-ray patent
834	6	Alive, x-ray patent
836	5 days	Myocardial infarction
837	2	Distemper, artery patent
840	6	Alive, x-ray patent
841	6	Alive, x-ray patent
844	14 days	Distemper, artery patent
845	6	Alive, x-ray patent
846	6	Alive, x-ray patent

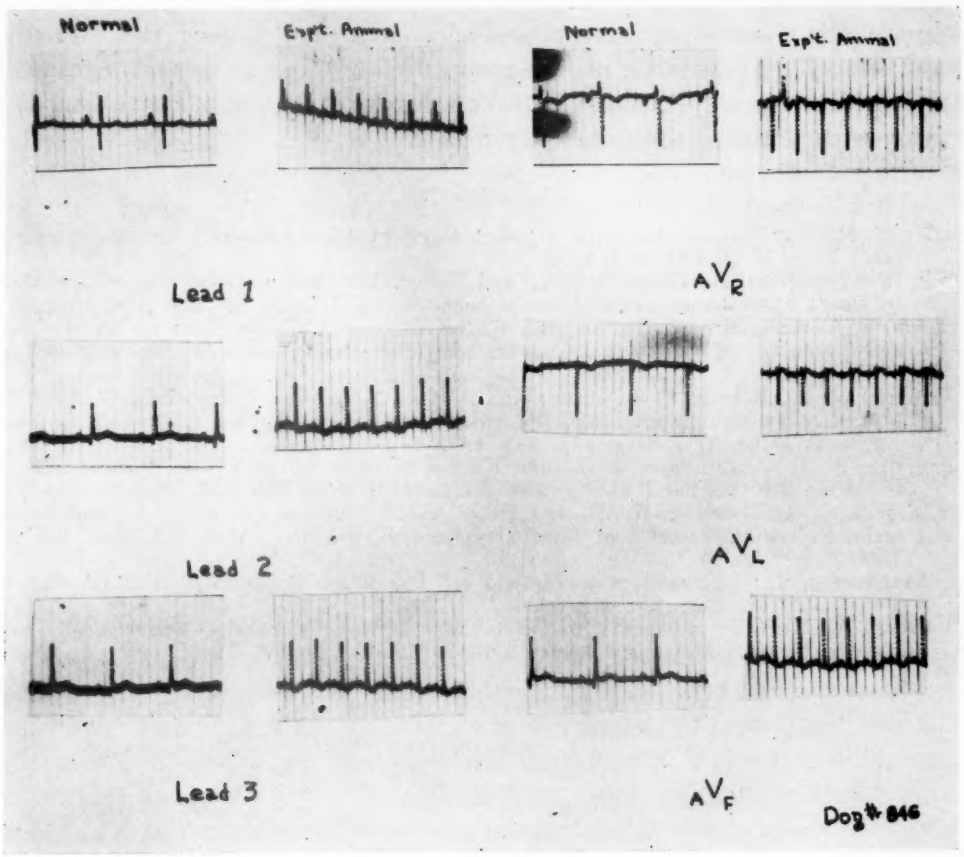


Fig. 6.—Postoperative electrocardiogram.

nutrition 3½ months and 1 month, respectively, after operation. Dogs 837 and 844 died of distemper 14 days and 2 months, respectively, postoperatively. Postmortem examination of these animals revealed no evidence of myocardial infarction or thrombosis at the arteriotomy site. In these 4 animals the circumflex coronary artery was completely patent. The 8 remaining animals are alive after 6 to 8 months. Coronary arteriograms have demonstrated that the circumflex coronary artery is patent (Figs. 4 and 5). Electrocardiographic tracings on these animals revealed no myocardial damage or coronary insufficiency. The electrocardiograms differ from those of normal animals only in an increase in heart rate (Fig. 6).

DISCUSSION

The internal diameter of the circumflex coronary artery of a dog is usually 2 to 4 mm. We have been discouraged by frequent early and late thrombosis of vessels of this size when direct anastomotic procedures are used. The demonstrated patency of this size vessel 6 months after sham coronary endarterectomy in this series is gratifying. The poor results obtained in the perfused group are not easily explained. Since thrombosis occurred only in segments of the vessel distal to the arteriotomy, it may have been due to an intimal reaction caused by the indwelling catheter.

SUMMARY

Sham coronary endarterectomy was carried out on two groups of dogs. In one group, distal coronary artery perfusion was used during the period of endarterectomy. All animals in this group died of thrombosis and myocardial infarction. In the nonperfused group two-thirds of the animals are long-term survivors with proved patent coronary vessels.

REFERENCES

1. Bailey, C. P., May, A., and Lemmon, W. M.: Survival After Coronary Endarterectomy in Man, J. A. M. A. **164**: 641, 1957.
2. Beck, C. S., Stanton, E., Batiuchok, W., and Leiter, E.: Revascularization of the Heart by Graft of Systemic Artery Into Coronary Sinus, J. A. M. A. **137**: 436, 1948.
3. Cannon, J. A., Longmire, W. P., Jr., and Kattus, A. A.: Considerations of the Rationale and Technique of Coronary Endarterectomy for Angina Pectoris, SURGERY **46**: 197, 1959.
4. Chardack, W. M., Bolgan, F. J., Olson, K. C., Gage, A. A., and Farnsworth, W. E.: The Mortality Following Ligation of the Anterior Descending Branch of the Left Coronary Artery in Dogs, Ann. Surg. **141**: 443, 1955.
5. Longmire, W. P., Jr., Cannon, J. A., and Kattus, A. A.: Direct-Vision Coronary Endarterectomy for Angina Pectoris, New England J. Med. **259**: 993, 1958.
6. McEachern, C. G., Manning, G. W., and Hall, G. E.: Sudden Occlusion of Coronary Arteries Following Removal of Cardio-respiratory Pathways, Arch. Int. Med. **65**: 661, 1940.
7. O'Shaughnessy, L.: Experimental Method of Providing Collateral Circulation to the Heart, Brit. J. Surg. **23**: 665, 1936.
8. Vineberg, A. M.: Development of Anastomosis Between Coronary Vessels and Transplanted Internal Mammary Artery, Canad. M. A. J. **55** 117, 1946.

WEDGE PULMONARY ARTERIOGRAPHY

Application in the Selection of Patients for Repair of Cardiac Defects

A. L. Loomis Bell, Jr., M.D., Seiichi Shimomura, M.D., Hugh F. Fitzpatrick, M.D., Charles F. Begg, M.D., and Harold A. Zintel, M.D.,
New York City, N. Y.

(From the Cardiopulmonary Laboratory, Department of Medicine and the Department of Surgery, St. Luke's Hospital)

THE importance of the pulmonary vasculature in regulating the circulation and, therefore, in determining the clinical course and operability of patients with congenital heart defects has been emphasized by the work of Edwards,[6] Dammann and Ferencz,[5] Heath and Edwards,[7] and Heath and others.[8, 9] Patients with large ventricular septal defects, widely patent ductus arteriosus, and other functionally related defects with elevated pulmonary artery pressures maintain a high pulmonary resistance early in life by persistence of the fetal pattern of thick medial hypertrophy of the small muscular pulmonary arteries and probably increased "vasomotor tone." Perhaps as early as the age of 2 years, in addition to muscular hypertrophy and increased tone, intimal proliferation appears, at first cellular and later fibrous. In contrast, patients with atrial septal defect and functionally related anomalies have reduced pulmonary resistance permitting large pulmonary flows through an abundant, dilated vascular bed at normal or near-normal pressures. Much later, usually in the third or fourth decade of life, intimal lesions may appear which are similar to those seen at an earlier age in patients with large ventricular septal defects.

These intimal lesions produce abrupt narrowing or occlusion of the pulmonary arteries and are associated with a generalized vascular dilatation, particularly of the small arteries and arterioles. When widely disseminated, these lesions rather than vasomotor tone appear to assume the major role in determining the resistance to blood flow through the lungs. At present these fibrous intimal occlusive changes are believed to represent a fixed and irreversible pathologic change which permanently reduces the functioning pulmonary vascular bed. Therefore, in a patient with increased pulmonary resistance presenting for surgical repair of the cardiac defect, it becomes of paramount importance to determine the nature of the pathologic changes responsible for the increased resistance.

Lung biopsy affords a direct method for assessment of the vascular lesions, but, because of the focal nature of the intimal lesions, in some instances the specimen studied may not reflect the over-all condition of the vascular bed unless serial slides are examined. Moreover, the dilated small arteries are not

This work was aided by a grant from the New York Heart Association.

Read at the Thirteenth Annual Meeting of the Society for Vascular Surgery, Atlantic City, N. J., June 7, 1959.

readily differentiated from normal vessels unless their relationship to the intimal lesions is recognized. Venous angiocardiograms and selective pulmonary arteriograms are useful in visualizing the main pulmonary arterial trunk and its major branches. However, these techniques do not provide selective filling of the small muscular arteries which are critically involved by the vascular lesions.

In previous reports, a clinical method for visualizing radiographically the small pulmonary arteries by wedge pulmonary arteriography (WPA) was presented.[1, 2, 4] Radiopaque dye, injected through a 2.5 mm. catheter which has been wedged in an equivalent size pulmonary artery, outlines the branches of this artery. Wedging of the catheter prevents rapid dissipation of the dye by blood flow, and the pattern of the small dye-filled vessels can be studied on the exposed arteriogram. This procedure has been used in the study of 65 cases of congenital or acquired heart disease.

The usefulness of the WPA in the selection of patients with congenital cardiac defects for surgical repair constitutes the substance of this report.

METHOD AND MATERIALS

During right heart catheterization a No. 7 Cournand catheter is manipulated into a pulmonary artery "wedge" position. Radiopaque dye (2 or 3 c.c.) is injected through the catheter at an injection pressure of approximately 100 mm. Hg. An x-ray exposure is made with an overhead tube on nonscreen film. Typical exposure factors are 110 kv., 300 Ma., at 1/30 second. The arteriograms so obtained result in a 1.1 to 1.2 times enlargement of the dye-filled vessels due to projection. For better definition, the film is enlarged approximately two times.

The patients studied by this technique fall into five main categories as shown in Table I. Patients with a "functional" heart murmur as the only manifestation to suggest heart disease have normal pulmonary artery pressure and blood flow. Patients with ventricular septal defect and functionally related defects have either a large defect with common pressures in aorta and pulmonary artery, or a smaller defect which creates a pressure gradient from aorta to pulmonary artery. Patients with large ventricular defects are further subdivided into the early-stage patients with muscular hypertrophy of the pulmonary arteries and the late-stage patients in whom occlusive intimal lesions are the predominant pathologic change. Most patients with atrial septal defects have increased pulmonary blood flow with

TABLE I. CLASSIFICATION OF PATIENTS STUDIED BY WEDGE PULMONARY ARTERIOGRAPHY

DIAGNOSIS	NUMBER OF PATIENTS
I. Normal	
"Functional" heart murmur only, normal pulmonary artery pressure, normal pulmonary blood flow	8
II. Ventricular septal defect and patent ductus arteriosus	
A. Large defects with common pressures in aorta and pulmonary artery	4
1. Early stage	
2. Late stage	
B. Smaller defects with higher pressure in aorta than pulmonary artery	5
III. Atrial septal defect	
A. Increased pulmonary blood flow with normal or near-normal pulmonary artery pressure	12
B. Decreased pulmonary blood flow with high pulmonary artery pressure	1
IV. Mitral stenosis	
Elevated pulmonary artery and left atrial pressures	21
V. Miscellaneous	14
Total	65

normal or near-normal pressures. However, one patient had an elevated pulmonary artery pressure with reduced pulmonary blood flow. Twenty-one patients with mitral stenosis of varying degrees of severity have been studied. The miscellaneous group includes patients having pulmonic stenosis with and without septal defects, multiple pulmonary emboli, congenital heart block, pericarditis, and fibroelastosis.

RESULTS

A typical normal wedge pulmonary arteriogram is shown in Fig. 1. The catheter is shown "wedged" at the bifurcation of a 2.5 mm. artery with dye outlining the abundant arborization of smaller arteries. Vessels as small as 100 micra are seen as discrete structures, with filling of the smaller arteries, arterioles, and capillaries producing a distinctive background pattern. Some dye has passed into the pulmonary veins. This 12-year-old girl has a "functional" heart murmur, with normal pulmonary artery pressure and flow.

Fig. 1.—V. L., 12-year-old girl with no heart disease, normal pressures, and normal pulmonary blood flow. This is a normal wedge pulmonary arteriogram. (See text for description.)

In contrast to the normal pattern, typical wedge pulmonary arteriograms of 2 patients from Group II with pulmonary hypertension, one due to a large ventricular septal defect and the other to truncus arteriosus are shown in Figs. 2 and 3. The arteriogram shown in Fig. 2 is from a 5-year-old boy with severe pulmonary hypertension due to ventricular septal defect and reversal of the shunt such that pulmonary flow is less than systemic flow. The appearance of wedge pulmonary arteriograms from this group is that of a reduced arborization pattern of the 2.5 mm. artery with abrupt termination of some arteries of 500 to 1,200 micra luminal diameter and sudden narrowing of other arteries into fine tortuous channels. The pattern of normal background filling is absent. A similar WPA is shown in Fig 3, in an older male with an inoperable cardiac defect. The late-stage changes of fibrous occlusive intimal and dilatation lesions

of the small pulmonary arteries associated with this WPA pattern are shown in Fig. 4, taken from the same case as Fig. 3.

The WPA of a patient with a small patent ductus arteriosus is shown in Fig. 5. Pulmonary artery pressure is only slightly elevated and the pulmonary blood flow exceeds systemic flow. The catheter has wedged at the bifurcation of a 2.5 mm. artery. Aborization of the branches is not as abundant as the normal pattern and reduced background filling is noted. Abrupt termination of the arteries, narrowed tortuous termination of arteries, and absent background filling as seen in late-stage patients is not seen. This WPA pattern of quantitative reduction in the arborization pattern and background filling is typical of patients with small defects or those with early-stage large ventricular defects. Histologic sections of the lung biopsy from the patient shown in Fig. 5 show medial hypertrophy of the muscular pulmonary arteries and arterioles (Fig. 6). No occlusive intimal lesions are demonstrated.

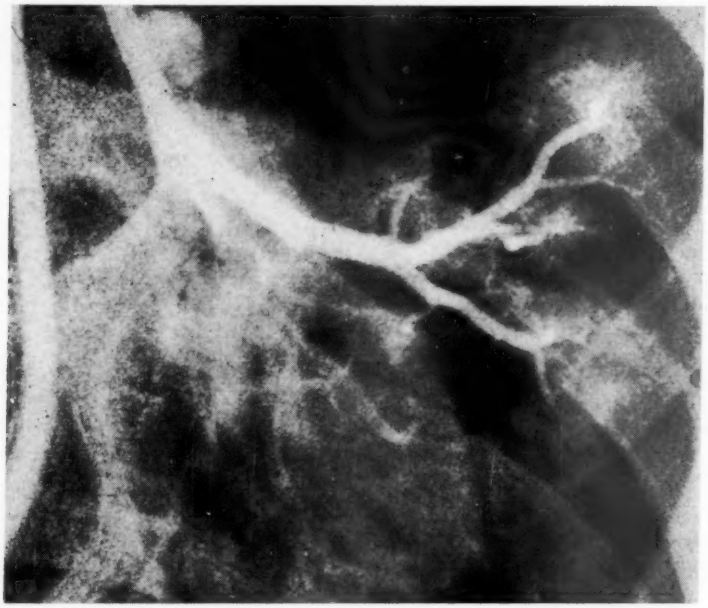


Fig. 2.—J. F. Wedge pulmonary arteriogram in a 5-year-old boy with a large ventricular septal defect. Pulmonary artery pressure 135/84, $\overline{98}$ mm. Hg. Pulmonary flow reduced to 80 per cent of systemic flow. Note sparse arborization, abrupt termination of some arteries, others narrow to fine, tortuous channels. There is lack of background filling. (See text for discussion.)

The WPA of a patient from Group III with an increased pulmonary blood flow due to atrial septal defect is shown in Fig. 7. In this patient the pulmonary flow is nearly five times normal with only slight increase in pulmonary artery pressure. The WPA shows an abundant arborization pattern with profuse background filling which exceeds normal. This pattern is correlated with the histologic sections taken from this patient at the time of operative repair of the defect and shown in Fig. 8.* The low-power section shows the typical widespread dilatation of various size pulmonary vessels giving a highly vascular appearance.

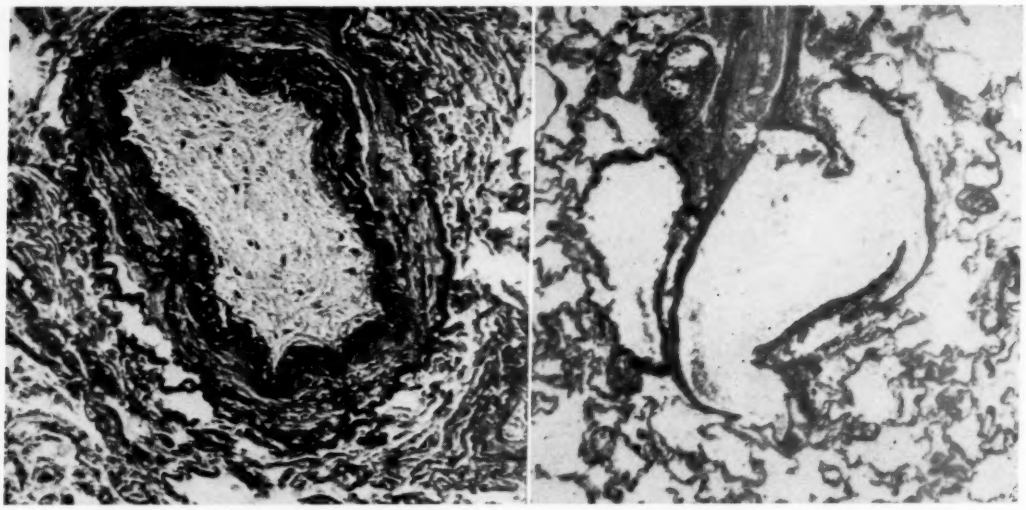
*Supplied by Dr. Henry T. Bahnson who closed the atrial septal defect of this patient.

One patient with atrial septal defect, pulmonary hypertension, and reversal of the shunt was studied (Fig. 9). This 45-year-old man experienced recent onset of cyanosis, easy fatigue, and breathlessness. On catheterization, pulmonary hypertension and reversal of the atrial shunt was demonstrated.

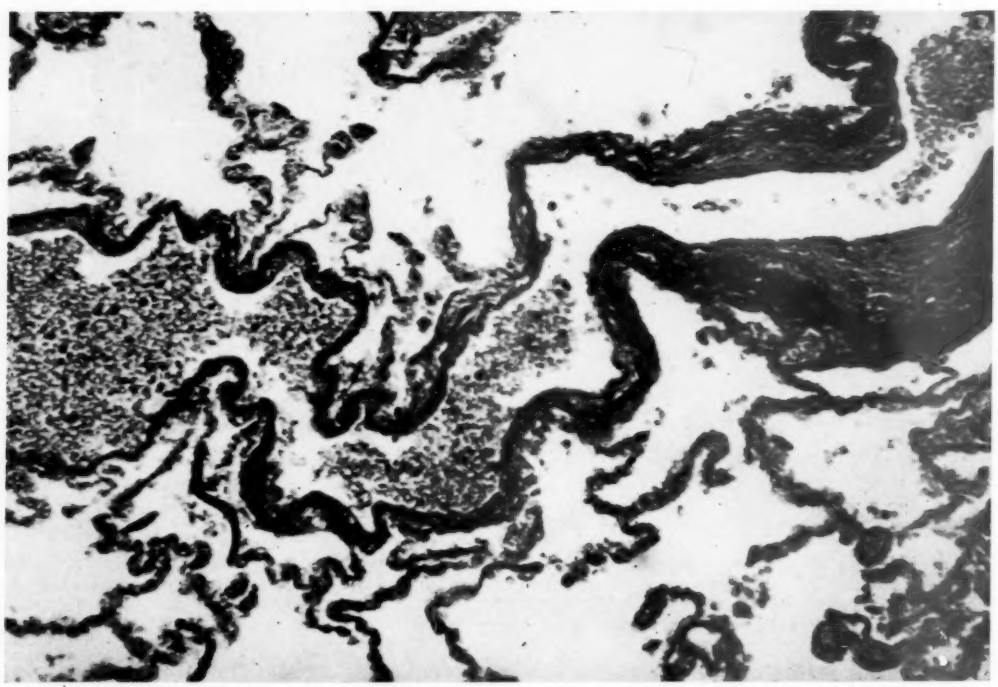
Fig. 3.—M. S., 18-year-old boy with truncus arteriosus. Pulmonary artery pressure 126/58, 86 mm. Hg. Pulmonary flow reduced to 63 per cent of systemic flow. Catheter tip unwedged (lies free in small pulmonary artery). Note sparse arborization and fine tortuous vessels without apparent filling of arterioles and capillaries.

The WPA of this patient reveals a reduced arborization pattern with narrowing of arteries into tortuous channels and without normal background filling. This WPA appearance is not unlike those patients with late-stage large ventricular septal defect and occlusive intimal lesions of 200 to 500 micra pulmonary arteries. Corrective surgery was believed hazardous for this patient and was not advised.

Patients with mitral stenosis and significant pulmonary hypertension show a reduced arborization pattern and background filling which bear an inverse relationship to the calculated pulmonary vascular resistance. A detailed report of these studies is being prepared and will be discussed elsewhere.[3] The largest



A. *B.*



C.

Fig. 4.—Histologic sections from M. S. Verhoeff–van Gieson stain. *A*, 280 micra pulmonary artery with medial hypertrophy and complete obliteration of lumen by fibrous intimal proliferation. *B*, Dilatation lesion in a nearly obstructed pulmonary artery of 250 micra size. *C*, Fibrous intimal thickening in a 150 micra pulmonary artery with thin-walled dilatation distally.

number of patients in the miscellaneous group have pulmonary stenosis which protects the small pulmonary arteries from the elevated right ventricular pressure. These patients have normal or reduced pulmonary flow. The WPA of this group of patients shows some narrowing of the arteries but is not differentiated with certainty from the normal.

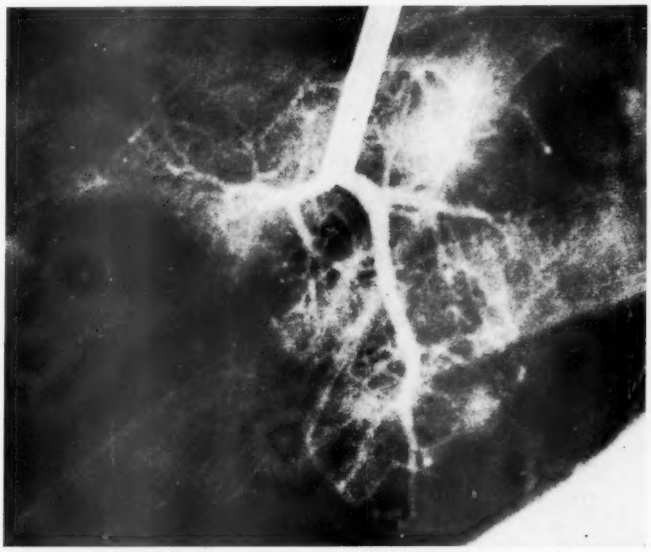
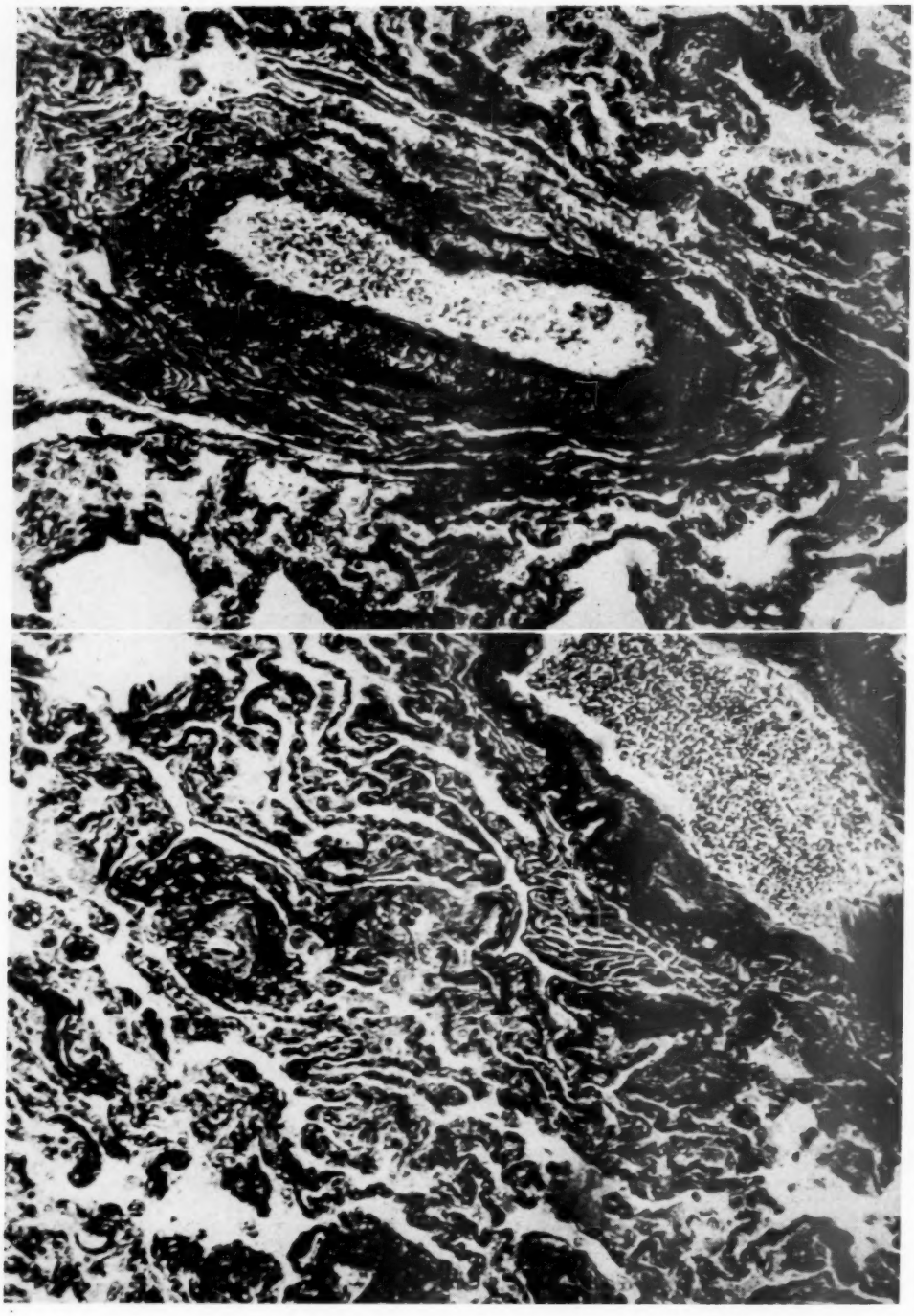


Fig. 5.—C. A., 31-year-old woman with patent ductus arteriosus. Pulmonary artery pressure 38/20, $\overline{27}$ mm. Hg. Pulmonary flow is 150 per cent of systemic flow. The catheter has wedged at the bifurcation of a 2.5 mm. artery. The arborization pattern and background filling are slightly reduced. (See text for description.)

DISCUSSION

In our experience to date, the wedge pulmonary arteriogram has provided an accurate clinical method for determining the type of the pathologic changes present in the pulmonary blood vessels. In patients with late-stage large ventricular septal defect, the WPA shows a distinctive pattern characterized by a reduced arborization, abrupt decrease in the caliber of 300 to 900 micra arteries, and absence of the normal background filling. In these cases, the pulmonary resistance is greatly increased and the pulmonary flow is usually reduced. Histologic sections of the lung specimens have demonstrated occlusive lesions of 300 to 900 micra arteries with dilatation lesions of smaller pulmonary arteries and arterioles. The pulmonary vascular bed is permanently reduced by these occlusive lesions, and, when the WPA pattern is characteristic, operative repair of the cardiac defect is considered to be contraindicated. This position has been strengthened by the course of a 4-year-old girl with a large patent ductus arteriosus, common aortic and pulmonary artery pressures, and an apparently "balanced" shunt. In one of the earliest wedge pulmonary arteriograms of the series, the above-described abnormal arborization pattern was encountered. Occlusion of the ductus at operation resulted in a substantial increase in pulmonary artery pressure and reduced aortic pressure. The patient died on the third postoperative day. Histologic sections showed advanced and disseminated occlusive intimal fibrosis.

A.



B

Fig. 6.—Histologic sections of lung biopsy from C. A. Verhoeff-van Gieson stain. *A*, Pulmonary artery of 145 micra size. Internal to external elastic lamina measures 33 micra, giving wall thickness of 23 per cent. *B*, Pulmonary arteriole of 55 micra size showing muscular hypertrophy. Pulmonary vein in upper right corner showing acellular intimal fibrosis considered to be an age change in this patient.

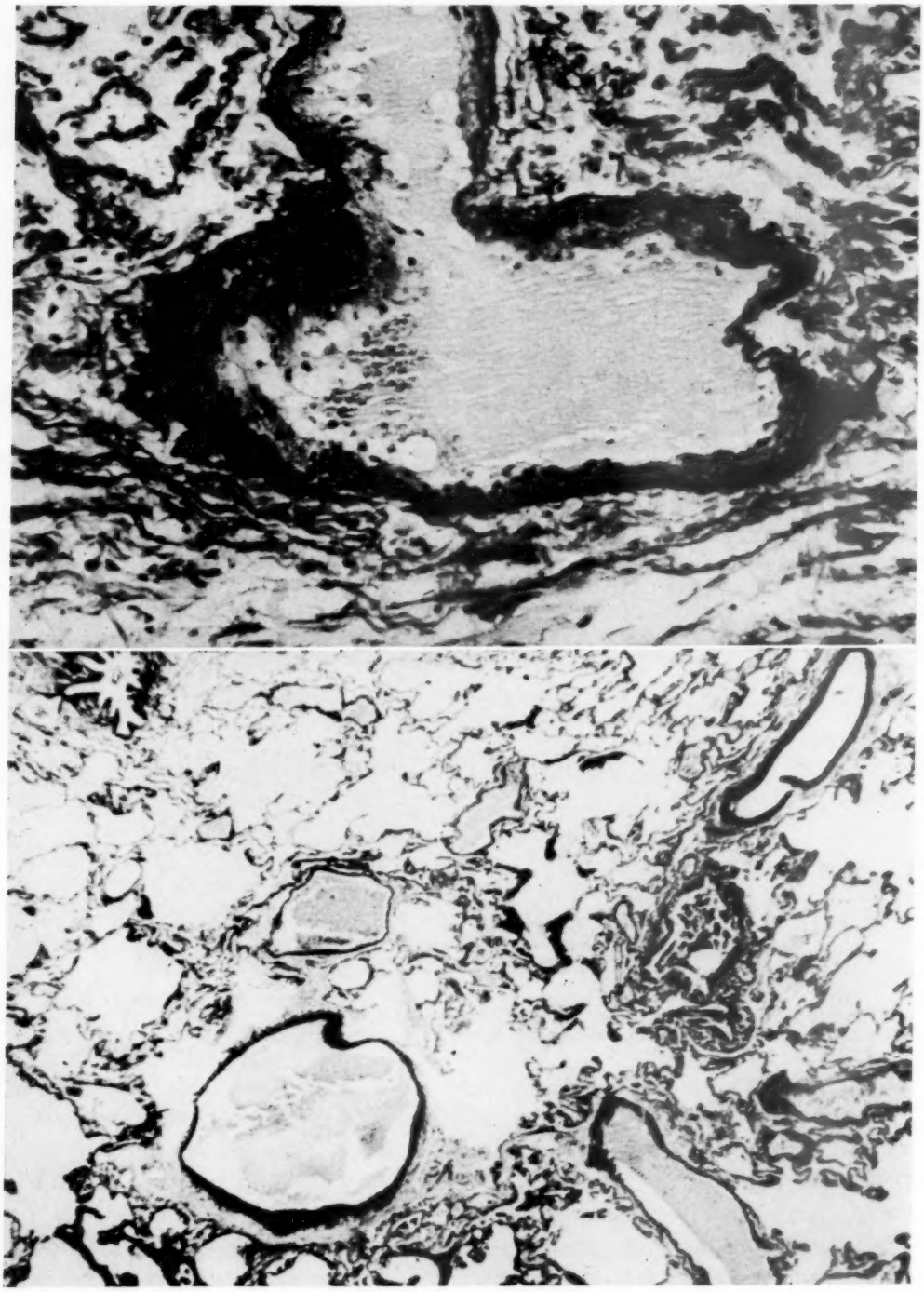
In early-stage large ventricular septal defect or where the defect is small and provides a protective resistance against left heart pressures and excessive pulmonary flow, the WPA pattern is one of reduced arborization and caliber of the branches and decreased but not absent background markings. In these cases, pulmonary flow usually exceeds systemic blood flow and the less severely increased pulmonary resistance apparently is maintained by medial hypertrophy and increased vasomotor tone, a reversible pathologic change. These patients are good-risk candidates for repair of the associated cardiac defect.

Fig. 7.—V. G., 34-year-old woman with an atrial septal defect. Pulmonary artery pressure 39/10, $\overline{16}$ mm. Hg. Pulmonary flow greatly increased to 470 per cent of systemic flow. Extensive arborization and abundant background filling are evident.

The WPA provides a reliable method for discriminating between the early-stage reversible and late-stage irreversible pathologic changes responsible for the increased resistance of these patients with large defects and common left and right ventricular pressures. Age alone is not a reliable criterion for differentiation, and hemodynamic data may be misleading especially in patent ductus arteriosus where incomplete mixing of pulmonary artery blood and differences in femoral and brachial arterial blood oxygen content make relative estimations of pulmonary and systemic blood flow uncertain.

Undoubtedly there are transitional cases between early- and late-stage pathologic changes where occlusive intimal lesions are present but less widespread and where vasomotor tone remains a critical determinant of pulmonary

A.



B.

Fig. 8.—Histologic sections from V. G. Verhoeff–van Gieson stain. *A*, Widespread dilatation of various sized pulmonary vessels. Vein in upper right-hand corner is 700 micra in diameter. *B*, Acellular intimal thickening of a 200 micra pulmonary vein.

resistance and, therefore, of the prognosis postoperatively. In these cases, clarification of the interrelationships between vasomotor tone and occlusive intimal lesions must await further studies. Of particular importance is the influence of a vasodilating agent such as acetylcholine on the pulmonary resistance of these cases.

In patients with atrial septal defect and increased pulmonary blood flow and normal pulmonary artery pressure, the WPA pattern is one of abundant arborization and background filling. In the 12 cases studied to date, the increased arborization pattern is proportional to the magnitude of the pulmonary blood flow. These patients are ideal candidates for operative repair of the defect.

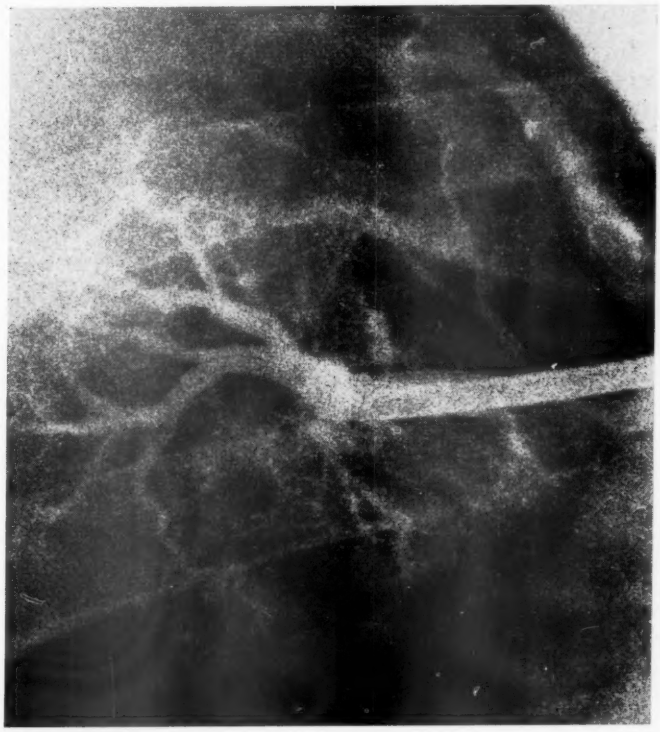


Fig. 9.—A. M., 45-year-old man with atrial septal defect. Pulmonary artery pressure is 73/63, 67 mm. Hg. Pulmonary flow is reduced to 70 per cent of systemic flow. Note similarity in pattern to J. F. in Fig. 2.

However, in late-stage atrial septal defect, occlusive intimal lesions occur in association with increased pulmonary resistance and reversed shunt.[6, 10] One such patient was encountered in our series and the WPA revealed the characteristic picture of reduced arborization and background filling typical of late-stage ventricular septal defect. Operative repair of the defect is considered contraindicated in this stage of the disease.

Untoward effects of the WPA procedure consist of the bitter taste of iodinated dye associated with cough in about one-half of the patients. Early in the series, 2 patients developed infarction of the wedged segment due to improper procedure.[2] This complication has been avoided by the subsequent use of smaller quantities of dye, standardized injection pressure, immediate flushing with saline, and a rapid procedure requiring a minimum period of wedging.

SUMMARY

Wedge pulmonary arteriography (WPA), a clinical method for visualizing the small pulmonary arteries radiographically, has been applied in 65 patients with cardiac defects. The pattern of the WPA is a reliable reflection of the pathologic changes present in the muscular pulmonary arteries.

When occlusive intimal lesions are present, the WPA appearance is distinctive, characterized by reduced arborization and abrupt termination of some arteries while others narrow to tortuous channels. The normal pattern of arteriolar and capillary filling is absent. When medial hypertrophy of the muscular arteries is present without intimal changes, the WPA shows reduced caliber of the arteries and less severe reduction of the arborization pattern and capillary filling. In patients with a dilated pulmonary vascular bed having increased pulmonary flow, with normal pulmonary pressures, the WPA is characterized by an overabundant arborization pattern and capillary filling proportional to the magnitude of the pulmonary blood flow. The significance of these WPA patterns in the selection of patients for repair of the associated cardiac defect is discussed.

We gratefully acknowledge the technical assistance of the Misses M. Mills, J. Wilbur, M. Guevera, and the Messrs. J. Davidse and F. Kleczek in the performance of this work. The valued cooperation of Dr. H. F. Hempel, Director of the Department of Radiology, has been essential to these studies.

REFERENCES

1. Bell, A. L. L., Jr., Shimomura, S., and Guthrie, W. G.: Wedge Pulmonary Arteriography: Application in Congenital and Acquired Heart Disease (Abstr.), Bull. New York Acad. Med. **34**: 763-764, 1958.
2. Bell, A. L. L., Jr., Shimomura, S., Guthrie, W. J., Hempel, H. F., Fitzpatrick, H. F., and Begg, C. F.: Wedge Pulmonary Arteriography: Application in Congenital and Acquired Heart Disease, Radiology **73**: 566, 1959.
3. Bell, A. L. L., Jr., Shimomura, S., and Taylor, J. A., Jr.: Observations on the Pulmonary Vascular Lesions of Mitral Stenosis as Studied by Wedge Pulmonary Arteriography. In preparation.
4. Bell, A. L. L., Jr., Shimomura, S., Taylor, J. A., Jr., and Fitzpatrick, H. F.: Detection of Pulmonary Lesions in Patients With Congenital and Acquired Heart Disease by Wedge Pulmonary Arteriography, Progress in Cardiovascular Diseases **2**: 64-75, 1959.
5. Dammann, J. F., Jr., and Ferencz, C.: Clinico-anatomic Correlations in Adjustments Between Systemic and Pulmonary Circulations, International Symposium on Cardiovascular Surgery, Philadelphia, 1955, W. B. Saunders Co., pp. 86-99.
6. Edwards, J. E.: Functional Pathology of the Pulmonary Vascular Tree in Congenital Cardiac Disease, Circulation **15**: 164-196, 1957.
7. Heath, D., and Edwards, J. E.: The Pathology of Hypertensive Pulmonary Vascular Disease, Circulation **18**: 533-547, 1958.
8. Heath, D., Helmholz, H. F., Jr., Burchell, H. B., DuShane, J. W., and Edwards, J. E.: Graded Pulmonary Vascular Changes and Hemodynamic Findings in Cases of Atrial and Ventricular Septal Defect and Patent Ductus Arteriosus, Circulation **18**: 1155-1166, 1958.
9. Heath, D., Helmholz, H. F., Jr., Burchell, H. B., DuShane, J. W., Kirklin, J. W., and Edwards, J. E.: Relation Between Structural Changes in the Small Pulmonary Arteries and the Immediate Reversibility of Pulmonary Hypertension Following Closure of Ventricular and Atrial Septal Defects, Circulation **18**: 1167-1174, 1958.
10. Heath, D., and Whitaker, W.: The Small Pulmonary Blood Vessels in Atrial Septal Defect, Brit. Heart J. **19**: 327-332, 1957.

(Society for Vascular Surgery papers continued in February issue.)